PretiFlaherty

Portland, ME
Augusta, ME
Concord, NH
Boston, MA
Washington, DC

Jeffrey D. Talbert
jtalbert@preti.com
207.791.3239

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Todd Kim
Assistant Attorney General
United States Department of Justice
Environment and Natural Resources Division
P.O. Box 7611
Washington, DC 20044-7611
pubcomment-ees.enrd@usdoj.gov

RE: Joint Comments on Proposed CERCLA Consent Decree Regarding the Diamond Alkali Superfund Site: *United States of America v. Alden Leeds, Inc., et al.*, Civil Action No. 2:22-cv-07326 (MCA-LDW) (D.N.J.); D.J. Ref. No. 90-11-3-07683/1

Dear Assistant Attorney General Kim:

The Small Parties Group ("SPG") respectfully submits these joint comments in support of the proposed CERCLA Consent Decree ("CD") lodged in *United States v. Alden Leeds, Inc., et al.*, No. 2:22-cv-07326 (MCA-LDW) (D.N.J.), regarding the Diamond Alkali Superfund Site ("Site").[1] *See* Notice of Lodging of Proposed Consent Decree Under the Comprehensive Environmental Response, Compensation, and Liability Act, 87 Fed. Reg. 78710 (Dec. 22, 2022), as amended by 88 Fed. Reg. 2133 (Jan. 12, 2023). The SPG includes (i) SPG parties that are parties to the CD ("the SPG Settling Parties"); (ii) Nokia of America Corporation, Pharmacia LLC, and Public Service Electric and Gas Company (PSEG), which participated in an underlying allocation but were not included in the settlement; and (iii) SPG parties that were not invited to participate in the allocation or the settlement (collectively, "Commenters").

This settlement is the culmination of over forty years of work by U.S. EPA on the Site and six years of intensive effort and arm's-length negotiation between U.S. EPA and eighty-five parties to advance the cleanup of the Lower Passaic River ("River") in Newark, New Jersey. Notwithstanding the constant efforts of Occidental Chemical Corporation ("OxyChem") (most recently through court filings and media advertisements) to avoid responsibility for cleaning up a river that it is primarily responsible for polluting with dioxin and other contaminants, this settlement is fair and reasonable, and the United States should promptly move for entry of the CD.

[1] The SPG Parties commenting are listed in Appendix 1, attached. These comments are also accompanied by a Table of Authorities listing all sources cited.

I. Background

In the 1980s, widespread dioxin contamination was discovered at 80/120 Lister Avenue in Newark, New Jersey ("Lister Facility"), the site of a former Agent Orange manufacturing plant located on the banks of the River.[2] The particular form of dioxin discharged from the Lister Facility—2,3,7,8 tetrachlorodibenzo-*p*-dioxin ("TCDD")—is the most toxic man-made substance known to exist.[3] The Governor of New Jersey declared a state of emergency,[4] and EPA placed the Site on its Superfund National Priorities List.[5]

One company—OxyChem—is responsible for the massive amounts of dioxin and other pollutants that were discharged from its former Agent Orange plant into the River. Indeed, the operation of OxyChem's Lister Facility resulted in one of the most significant mass contributions of TCDD to a United States river ever recorded.[6] It is also well documented that the Lister Facility discharges were made knowingly and recklessly, threatening worker safety and making the River one of the most dioxin-contaminated rivers in the world.[7] The dioxin and other contaminants discharged from the Lister Facility spread through the River and beyond via tidal action and hydrodynamic forces in the estuarine system.[8]

In 2016, after years of extensive studies and numerous targeted cleanup actions, EPA issued a Record of Decision selecting the final remedy for the contaminated sediments in Operable Unit 2 ("OU2") of the Site, comprising the lower 8.3 miles of the River.[9] Although EPA identified eight contaminants of concern ("COCs") in OU2, the selected remedy is primarily aimed at

[2] U.S. E.P.A., Record of Decision: Lower 8.3 Miles of the Lower Passaic River Part of the Diamond Alkali Superfund Site at 3 (Mar. 3, 2016), semspub.epa.gov/src/document/02/396055 ("2016 ROD").

[3] *See* Linda S. Birnbaum, *Report* (Mar. 21, 2023) at 2, 8, attached as Exhibit A; N.J. Dep't of Env't Prot., Memo Re: Lower Passaic River - Phase I Removal Action Surface Water Discharge Permit Equivalent, at 3-4 (Dec. 10, 2010) ("NJDEP Memo"); Richard F. Bopp et al., *A Major Incident of Dioxin Contamination: Sediments of New Jersey Estuaries*, 25 ENV'T. SCI. & TECH. 951, 951 (1991); 2016 ROD at 3-4. Unless otherwise specified, all references to dioxin in these comments refer to TCDD.

[4] N.J. Exec. Order No. 40 (June 2, 1983).

[5] Amendment to National Oil and Hazardous Substance Contingency Plan, National Priorities List, 49 Fed. Reg. 37070 (Sept. 21, 1984) (codified at 40 C.F.R. Part 300).

[6] Bopp, *supra* note 3, at 955.

[7] *See id.*; *Diamond Shamrock Chems. Co. v. Aetna Cas. & Sur. Co.*, 609 A.2d 440, 446-49, 454-55, 461-64 (N.J. Super. App. Div. 1992) ("*Aetna II*").

[8] 2016 ROD at 12, 16.

[9] *See generally* 2016 ROD.

addressing risks from one of those COCs: dioxin.[10] The risks to human health and the environment from dioxin far exceed those from any other contaminant in the River.[11]

After identifying a list of potentially responsible parties ("PRPs") for OU2,[12] EPA exercised its substantial discretion to convene a "fair, carefully structured, information-based allocation" to determine the proper distribution of relative responsibility for remediation costs;[13] it selected a qualified third-party neutral—AlterEcho—to complete the allocation ("Allocator");[14] and it invited certain of the identified PRPs ("Allocation Parties") to participate in the allocation.[15]

Most of the Allocation Parties agreed to participate; one notable exception was OxyChem, which refused to partake in the allocation[16] and instead initiated costly litigation against scores of companies whose contributions, if any, to the contamination in the River were infinitesimal in comparison to OxyChem's.[17] The transparent purpose of OxyChem's Litigation was to disrupt EPA's cooperative settlement process and to impose inordinate costs on the cooperating parties. Indeed, OxyChem continues with this agenda even now, having launched a deceptive public relations campaign aimed at instilling fear in the residents of New Jersey by misleading the public

[10] *See id.* at 1-2, 41, 89.

[11] *See* Yeh Decl. ¶ 8, ECF No. 84-1 in *Alden Leeds*.

[12] Letter from Nicoletta Di Forte, EPA, to PRPs (Mar. 31, 2016).

[13] Letter from Eric J. Wilson, EPA, to Marcia E. Backus, Occidental Petroleum Corp. (Nov. 28, 2017), ECF No. 2246-6 in OxyChem's Litigation.

[14] Letter from Eric J. Wilson, EPA, to OU2 PRPs at 1 (Sept. 18, 2017), Att. A to AlterEcho, Final Allocation Recommendation Report (Dec. 28, 2020) ("Allocation Report"). The Allocation Report and all its attachments are available at semspub.epa.gov/src/document/02/609904.

[15] Letter from Eric J. Wilson, *supra* note 14, at 1; *see* AlterEcho, Diamond Alkali Superfund Site OU2 Allocation Guide § 3 (June 15, 2018, as amended, July 6, 2018, and Mar. 22, 2019), Att. G to Allocation Report ("Allocation Guide").

[16] *See* List of Participating Allocation Parties, Att. I to Allocation Report.

[17] *See Occidental Chem. Corp. v. 21st Century Fox Am., Inc.*, No. 2:18-cv-11273-MCA-LDW (D.N.J.) ("OxyChem's Litigation"). OxyChem's Litigation has been stayed pending disposition of the *Alden Leeds* matter. ECF No. 2287 in OxyChem's Litigation, as amended by ECF No. 2289. OxyChem's preference for litigation over cooperation has been made clear in other matters as well. When the State of New Jersey levied related claims against OxyChem and others under the New Jersey Spill Act, OxyChem was the very last party to settle those claims. *See N.J. Dep't of Env't Prot. v. Occidental Chem. Corp.*, No. ESX-L9868-05 ("Spill Act Litigation"), Consent Judgment and Order on the Entry and Approval of the Consent Judgment (N.J. Super. Ct. Law Div. Dec. 16, 2014), ECF Nos. 951-30, 951-31 in OxyChem's Litigation. OxyChem also spent ten years fighting the federal government in court in an effort to skirt its responsibility for another infamous dioxin site, the Love Canal. *See United States v. Hooker Chems. & Plastics Corp.*, 680 F. Supp. 546 (W.D.N.Y. 1988); *United States v. Hooker Chems. & Plastics Corp.*, 722 F. Supp. 960 (W.D.N.Y. 1989); U.S. E.P.A. Press Release, *Occidental Chemical Signs Consent Order for Storage and Destruction of Love Canal Wastes* (June 1, 1989), epa.gov/archive/epa/aboutepa/occidental-chemical-signs-consent-order-storage-and-destruction-love-canal-wastes.html.

into believing that they, not OxyChem, will bear the remaining River cleanup costs if the CD is approved.

The parties that participated in the allocation devoted nearly four years and extensive resources to complying with a detailed allocation process developed and approved by EPA. The SPG Settling Parties, Nokia, Pharmacia, and PSEG compiled and submitted to the Allocator materials requested by EPA or the Allocator, responded to detailed information requests regarding their facilities, provided technical information, and executed certifications to the completeness of their submissions.[18] If OxyChem had participated in the allocation, it could have raised any arguments it had about the methodology, the information submissions, or the results of the process.

In December 2020, the Allocator issued the Final Allocation Recommendation Report, recommending each Allocation Party's relative share of responsibility for OU2 Site costs based on the allocation methodology approved by EPA.[19] The Allocator found that one Allocation Party—OxyChem—contributed almost all of the TCDD to the Site.[20] Thus, the Allocator concluded that OxyChem alone should bear responsibility for paying more than 99.9% of the costs

[18] Allocation Guide §§ 4.1, 4.5.

[19] *See generally* Allocation Report.

[20] *Id.* at 19.

to remediate the Site.[21] In contrast, the Allocator determined that the contributions of each of the other Allocation Parties could be measured by fractions of a percent.[22]

The Allocation Report was used and considered by EPA in settlement negotiations with the Settling Parties as to OU2 as well as Operable Unit 4 ("OU4"), which comprises the lower seventeen miles of the River: the lower 8.3 miles (OU2) plus the upper 9 miles above OU2.[23] The results of those negotiations are embodied in the CD that the United States lodged with the Court on December 16, 2022.[24]

OxyChem engaged a cadre of hired experts and public relations firms to attack the settlement in the press, but the reality is that the proposed CD is not breaking new ground. It follows a well-worn path forged by many prior settlements between the United States and parties with demonstrably small contributions to the contamination of a Superfund Site. The proposed settlement is a well-supported, fair, and reasonable resolution of the SPG Settling Parties' responsibility for what is fundamentally a one-COC, one-party Site. Although a detailed analysis

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[21] Allocation Operations Spreadsheet, Att. K to Allocation Report. The Allocator also provided an "alternative" calculation for consideration in "settlement efforts." Allocation Report at 28-29, 35. The Allocator did not recommend the alternative calculation, which distributes orphan shares to Allocation Parties by COC as opposed to pro rata, and the Allocator acknowledged that the alternative calculation is contrary to the approved Allocation Protocol and is inconsistent with case law regarding the "traditional methodology for redistribution of the orphan share." Allocation Report at 28; *cf.* AlterEcho, Diamond Alkali Superfund Site OU2 Allocation Protocol at 2, Att. H to Allocation Report ("Allocation Protocol"); *see, e.g.*, *Action Mfg. Co. v. Simon Wrecking Co.*, 428 F. Supp. 2d 288, 328-29 (E.D. Pa. 2006), *aff'd*, 287 F. App'x 171 (3d Cir. 2008) (stating that "the other federal courts of appeals to consider the issue have concluded or assumed that the orphan shares should be allocated equitably among plaintiff and defendant PRPs" and allocating orphan shares among parties "in proportion to their relative liability at the Site" because this was the "fairest way to divide it"); *Caldwell Trucking PRP Grp. v. Pullman Co.,* No. 95-1690 (DMC), 2002 U.S. Dist. LEXIS 28410, at *81, 84 (D.N.J. Nov. 21, 2002), *aff'd sub nom. Caldwell Trucking PRP v. Rexon Tech. Corp.*, 421 F.3d 234 (3d Cir. 2005) ("Equity demands that all of the parties bear proportionate responsibility for any 'orphan share.'. . . The Court holds that any calculated orphan share should be apportioned *pro rata* according to the parties' allocation shares of liability."); *United States v. Kramer*, 953 F. Supp. 592, 598 (D.N.J. 1997) ("It appears much more equitable to apportion the orphan shares to all the [parties] according to their relative equitable share." (quotation marks omitted)); *Litgo N.J., Inc. v. Martin*, No. 06-2891 (AET), 2011 WL 65933, at *5 (D.N.J. Jan. 7, 2011) ("[T]he Court believes that a pro rata allocation of any orphan share assigned to Columbia Aircraft would be most appropriate . . . ."). OxyChem should be responsible for the vast majority of the orphan share (primarily from unrepresented shares from entities such as the Passaic Valley Sewerage Commission ("PVSC") and the municipalities that were excluded from the allocation by EPA versus other PRPs. Under the "alternative" calculation, the opposite occurred. Even the Allocator noted that "this alternative approach results in the potentially inequitable result of a higher overall share of responsibility, for a remedy primarily driven by risks associated with the presence of dioxin/furans, being assigned to Allocation Parties that constitute a relatively small number of parties that contributed a lower risk COC." Allocation Report at 29.

[22] Allocation Operations Spreadsheet, Att. K to Allocation Report.

[23] *See* CD at 5-6, ECF No. 2-1 in *Alden Leeds*; Allocation Guide §§ 3.1, 7.2(a); *see* Notice of Lodging, 87 Fed. Reg. 78710 (noting that the settlement was "[b]ased on the results of the allocation").

[24] ECF Nos. 2, 2-1 in *Alden Leeds*.

of the technical underpinning of the settlement is unnecessary to support judicial approval of the CD, and courts should defer to EPA's expertise on such matters,[25] the underlying bases of the settlement also followed generally-accepted scientific principles applied to the historical facts of the Site.[26] Given that the Settling Parties are paying a significant premium over their *de minimis* responsibility, the settlement easily falls within the United States' broad statutory discretion to resolve CERCLA claims. The settlement would also provide significant funds that EPA can put toward cleanup efforts in the near term, rather than forcing EPA to have to wait for years of litigation to play out.

In support of the settlement, these comments address the risk posed by dioxins at the Site, *see infra* Part II.A, and the reasons for including OU4 within the settlement, *see infra* Part II.B. These comments also discuss OxyChem's long and well-documented history of discharging dioxin and other COCs directly into the River. *See infra* Part III. Finally, the comments address the very significant premium that EPA applied to the settlement, by which the Settling Parties will be paying hundreds of times more than their allocated responsibility, and some factors that were weighed to benefit OxyChem that further increase the premium and leave little doubt about the fairness and reasonableness of the settlement. *See infra* Part IV.

For the reasons set forth in these comments, the United States should continue to support the settlement and submit a motion requesting entry of the CD in the District Court in the *Alden Leeds* matter, as well as engage in good faith settlement negotiations with the remaining participating allocation parties and the remaining defendants and third-party defendants in OxyChem's Litigation.

II. The Diamond Alkali Superfund Site is Overwhelmingly a Dioxin Site.

Dioxins and furans are by far the most toxic of the contaminants found at the Site.[27] In particular, TCDD is the most toxic man-made substance known to exist and therefore dominates the risks posed by the Site.[28] For perspective, TCDD is ten times more toxic than the most toxic

[25] *See United States v. Cannons Eng'g Corp.*, 899 F.2d 79, 88 (1st Cir. 1990).

[26] *See, e.g.*, Exh. A; D. Michael Johns, *Relative Risks of OU2 Contaminants of Concern & Comparison with OU4: A Summary of Data Information Regarding Relative Ecological Risks for the Lower Passaic River* (Mar. 22, 2023), attached as Exhibit B; Betsy Ruffle, *Relative Risks of OU2 Contaminants of Concern & Comparison with OU4: A Summary of Data and Information Regarding Relative Human Health Risks for the Lower Passaic River* (Mar. 22, 2023), attached as Exhibit C.

[27] 2016 ROD at 28-29, 33; Yeh Decl. ¶ 8; Allocation Protocol, Att. A at 16-19.

[28] NJDEP Memo, *supra* note 3, at 3 (stating that TCDD is "considered among the most toxic synthetic chemicals known to humankind"); Bopp, *supra* note 3, at 951 (stating that TCDD is "among the most toxic chemicals ever tested"); U.S. E.P.A., *Learn About Dioxin*, www.epa.gov/dioxin/learn-about-dioxin (last visited Mar. 15, 2023) (describing dioxins as "highly toxic"); Exh. A at 2-3 (explaining that TCDD is the benchmark used to measure the toxicity of all other dioxin-like substances).

polychlorinated biphenyl ("PCB") congener, PCB-126, and *many thousands of times more toxic than the average PCB*.[29]

TCDD poses a significant threat to human health even at very low levels of exposure.[30] TCDD can cause cancer in multiple parts of the body, and it can also cause a host of non-cancer health issues, including cardiovascular disease, diabetes, endometriosis, early menopause, altered immunologic response, altered growth factor signaling, and altered metabolism.[31] Exposure of infants, children, and young people to TCDD is of particular concern because it can result in multigenerational effects such as altered thyroid and immune status, neurobehavior, cognition, development of reproductive organs, and sex ratios of offspring.[32]

A. Because TCDD poses a far greater risk than other contaminants at the Site, the COC risk ranking in the allocation is appropriate.

Due to its extreme toxicity, TCDD has been the primary COC at the Site since its discovery there in 1983.[33] Since that time, TCDD has—appropriately—remained a focus of the remedies selected by EPA for OU2 and OU4 and through the allocation and settlement itself.[34]

In the 2016 ROD for OU2, EPA identified the eight COCs that it found to "pose the greatest potential risks to human health and the environment."[35] Of those eight, EPA concluded that dioxins and furans pose the greatest risk—in some cases, by several orders of magnitude.[36] EPA

[29] Exh. A at 3, 8.

[30] *Id.* at 2, 4, 7-8; *see* U.S. E.P.A., Record of Decision for an Interim Remedy in the Upper 9 Miles of the Lower Passaic River Study Area, OU4 of the Diamond Alkali Superfund Site at 8 (Sept. 28, 2021), semspub.epa.gov/src/collection/02/AR65669 ("2021 ROD").

[31] Exh. A at 3-8; *see* 2016 ROD at 15.

[32] Exh. A at 7-8; *see* 2016 ROD at 15; 2021 ROD at 15.

[33] *See* N.J. Exec. Order No. 40; National Priorities List, 49 Fed. Reg. 37070.

[34] Yeh Decl. ¶ 8; 2016 ROD at 14-15, 29-30; 2021 ROD at 7, 15, 24-25, 28-36; Allocation Protocol at 6, Att. A at 16-19; U.S. E.P.A., Memo. Re: Request for Authorization to Conduct a CERCLA Non-Time-Critical Removal Action at the Diamond Alkali Site, Newark, Essex County, New Jersey, at 5 (Jan. 8, 2009), semspub.epa.gov/src/document/02/239617 ("Request for Authorization"); CD at 3, 5.

[35] 2016 ROD at 14-16, 21, 43-44; *see* 42 U.S.C.A. § 9602(a) (authorizing EPA to regulate hazardous substances that "when released into the environment may present substantial danger to the public health or welfare or the environment").

[36] *See* 2016 ROD at 29-30; Yeh Decl. ¶ 8; Request for Authorization at 5 ("EPA and NJDEP believe these sediments pose a serious threat, because their dioxin concentrations are well over three orders of magnitude greater than the average surface sediment dioxin concentrations in the rest of the River (0.8 ppb), and their highly toxic concentrations would pose significant risk to human health or the environment should exposure occur.").

selected a bank-to-bank capping remedy for OU2 that focused on achieving remediation goals based primarily on risk to human health from TCDD.[37]

The baseline human health risk assessment ("HHRA") and ecological risk assessment ("ERA") performed for OU2 support this remedy.[38] A baseline HHRA and ERA also showed that TCDD poses the greatest risk to human health and the environment in OU2 and OU4, further supporting remedial action to address TCDD contamination.[39]

Indeed, based on risk assessments performed for the lower seventeen miles of the Passaic River, TCDD contributes nearly all the risk to human health and the environment in the River.[40] Therefore, in the Allocation, EPA appropriately recognized that TCDD poses the greatest human and ecological risk and drives the need to remove and cap sediments in the River.[41] EPA also appropriately targeted dioxin contamination in order to protect human health and the environment through dredging and capping remedies in the upper nine miles of OU4.[42]

For these reasons, and with EPA's approval, the Allocator determined each Allocation Party's "contribution of each COC in OU2 sediments, as weighted by the relative risk each COC poses to human health and the environment."[43] In deciding to use a risk-weighted approach, the

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[37] 2016 ROD at 12, 41-45, 57-58, 85, 89.

[38] *Id.* at 41; The Louis Berger Group et al., *Lower Eight Miles of the Lower Passaic River: Remedial Investigation Report for Focused Feasibility Study*, at 1-31 to 1-33, 2-8 to 2-11, 4-41 to 4-43 (Mar. 3, 2014), semspub.epa.gov/src/document/02/703639 ("OU2 FFS"); Human Health Risk Assessment, App. D of the OU2 FFS, at 6-1 to 6-6 semspub.epa.gov/src/document/02/703642; AECOM, *Baseline Human Health Risk Assessment for the Lower Passaic River Study Area*, at ES-13 to ES-16 (July 2017), semspub.epa.gov/work/02/616138.pdf; Windward Env't LLC, *Lower Passaic River Study Area Baseline Ecological Risk Assessment*, at ES-35 (June 17, 2019), semspub.epa.gov/work/02/620400.pdf. These risk assessments were performed for EPA's OU2 FFS and the 2016 ROD and used by EPA in the 2016 ROD. 2016 ROD at 6, 21.

[39] *See* Anchor QEA, LLC, *Remedial Investigation Report: Lower Passaic River Study Area Remedial Investigation/Feasibility Study* at 172-74, 220-21 (July 2019), ourpassaic.org/RemedialInvestigation.aspx ("Lower 17-Mile RI Report"); Exh. C at 3-2, 3-5 to 3-7; Exh. B at 7, 14-15. These risk assessments used site data collected during the seventeen-mile remedial investigation to determine the risks to human and ecological receptors who may be exposed to contaminants of concern in the lower seventeen miles of the river. EPA approved the HHRA in July 2017 and the BERA in June 2019. 2021 ROD at 6.

[40] *See* Yeh Decl. ¶ 8 (stating that dioxins/furans are responsible for 80-90% of the overall relative risk in OU2); Lower 17-Mile RI Report at 172-74, 220-21; Allocation Protocol, Att. A at 16-19; Exh. C at 3-5 to 3-7 (explaining that the relative contribution of TCDD to total incremental human health risk is between approximately 75% and 96%, depending upon the risk scenario evaluated, and stating that TCDD poses nearly all of the risk to human health from consumption of fish and crab); Exh. B at 14-15 (showing that, for the River, TCDD poses nearly all of the risk to ecological receptors (i.e., invertebrates, fish, and mammals) in OU2 and OU4).

[41] *See* Letter from Eric J. Wilson, *supra* note 14.

[42] 2021 ROD at 36, 52-53; *accord id.* at App. 4.

[43] Allocation Report at 18-19; *see* Allocation Protocol at 2.

Allocator specifically noted "*the extreme differential in risk created by the release of one of the COCs, dioxin/furans, almost exclusively by one of the Allocation Parties,* [*OxyChem*]*, in relation to that of the other COCs" at the Site*.[44] Such a risk-weighting is embraced by CERCLA;[45] it is generally accepted in the scientific field;[46] it has been endorsed by EPA[47] and courts[48] alike; and it supports EPA's regulatory assessment of this Site, pursuant to which EPA selected a bank-to-bank remedy for OU2 that is primarily driven by dioxin.[49]

The risk ranking and ultimate risk percentages used by the Allocator incorporate the data and information that EPA used to characterize human health in the OU2 FFS and the 2016 ROD.[50] That data and information is based on human exposure to the eight COCs through ingestion of the fish and crab found in the relevant part of the River.[51] The Allocator's final result—assigning dioxin 83.92% of overall relative risk—is consistent with the cancer and noncancer human health

[44] Allocation Report at 19 (emphasis added).

[45] *See* 42 U.S.C.A. § 9621(d)(1) (requiring EPA to select remedial action that "assures protection of human health and the environment").

[46] *See* Travis R. Kline, *Redefining Environmental Cost Allocation Using the Toxicity Factor*, alterecho.com/artfiledownload/8/CostAllocationTravisKline.pdf (last visited Mar. 17, 2023) (setting out the Toxicity Adjusted Paradigm approach, which accounts for the concentrations and toxicity of each COC and potential for exposure and noting that "[c]orrective action, as practiced under EPA and most state policies and programs, is heavily influenced by risk assessment").

[47] *See* U.S. E.P.A., Memo. Re: Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions, Off. of Solid Waste and Emergency Response Directive 9355.0-30 (Apr. 22, 1991), nepis.epa.gov/exe/zypurl.cgi?dockey =910165cr.txt (setting out EPA's remedial decision-making process under the National Contingency Plan, which equally considers human cancer risk, human noncancer hazard, and ecological hazard); Toxic and Hazardous Substances Control - Contractor and Subcontractor Access to Confidential Business Information, 52 Fed. Reg. 19921 (May 28, 1987) (discussing EPA's role in determining whether "disposal of certain chemical substances or mixtures may present an unreasonable risk of injury to human health or the environment"); Notice of Request for Public Comment, 50 Fed. Reg. 5034, 5037 (Feb. 5, 1985) ("If a waste contributed by one or more of the parties offering a settlement disproportionately increases the costs of cleanup at the site, it may be appropriate for parties contributing such waste to bear a larger percentage of cleanup costs than would be the case by using solely a volumetric basis.").

[48] *See AlliedSignal, Inc. v. Amcast Int'l Corp.*, 177 F. Supp. 2d 713, 724, 752 (S.D. Ohio 2001) (focusing an allocation of costs on mass and toxicity and noting that "[c]ourts have held that it is appropriate to allocate a greater share of responsibility to a party whose waste is more toxic"); *New York v. Solvent Chem. Co.*, 685 F. Supp. 2d 357, 385, 428-29, 451 (W.D.N.Y. 2010), *vacated and remanded in part on other grounds*, 453 F. App'x 42 (2d Cir. 2011) (adopting a "risk-weighted" allocation method, referred to as the "relative toxicity risk assessment"); *cf. Trinity Indus., Inc. v. Greenlease Holding Co.*, 903 F.3d 333, 358 (3d Cir. 2018) (stating that a volumetric approach is "divorced from record evidence and analytically unsound" when it fails to adequately account for equitable considerations particular to a site).

[49] *See* 2016 ROD at 1-2, 41, 89.

[50] *See* OU2 FFS at 1-31 to 1-33.

[51] Exh. C at 3-1 to 3-2.

risks posed by dioxins in comparison to the other seven COCs.[52] If considered alone, the cancer human health risk posed by dioxin would be 92.24% of the relative risk and the noncancer human health risk would assign dioxin 81.99% of the relative risk.[53] When combined with ecological relative risk percentages,[54] a final relative risk percentage of 83.92% for dioxin is reasonable and appropriate.

B. EPA reasonably included OU4 in the settlement.

Armed with abundant fate and transport and risk-related Site data, it was appropriate for EPA to extend the Allocation results for OU2 to include the upper nine miles of OU4 in the settlement agreement.[55] The remedies in those operable units will complement one another,[56] and it is well documented that contaminants in the River are mixed and distributed through normal river flow dynamics and tidal influences (including but not limited to the so-called estuarine turbidity maximum or "salt wedge") from river mile zero (bottom of OU2 and OU4) to the Dundee Dam (top of OU4).[57]

In addition, the risk assessments performed for the River support extending the Allocation and settlement agreement to OU4. Those risk assessments—both human health and ecological—demonstrate that the risk drivers, toxicity of COCs, and exposure pathways in OU2 and OU4 are substantially similar.[58]

Specifically, the human health risks that EPA evaluated for OU2 are substantially similar to those identified in the OU4 RI/FS.[59] EPA concluded for both OU2 and OU4 that consumption of fish and crab are the key exposure pathways associated with the highest cancer risks and

[52] Allocation Protocol, Att. A at 18.

[53] *Id.* at 17; Exh. C at 3-5.

[54] Allocation Protocol, Att. A at 18.

[55] *See* CD at 5-6.

[56] *See id.* at 6.

[57] Lower 17-Mile RI Report at 17-25; 2016 ROD at 16-17.

[58] *Compare* OU2 FFS at 1-31 to 1-33 *with* Lower 17-Mile RI Report at 162-71, 173, 175-95, 220-21 *and* Integral Consulting Inc., *Upper 9-Mile Source Control Interim Remedy Feasibility Study, Lower Passaic River Study Area Remedial Investigation and Feasibility Study* at 2-10 to 2-11 (Sept. 9, 2021), semspub.epa.gov/work/02/625207.pdf ("Upper 9-Mile IR FS"); Yeh Decl. ¶ 8 (explaining that the risk drivers in OU2 and OU4 are the same); *see* 2016 ROD at 22-41; 2021 ROD at 23-36; Exh. C at 3-6 to 3-7 (comparing risks in OU2 and OU4); Exh. B at 13-15 (same).

[59] *See* Upper 9-Mile IR FS at 2-11; OU2 FFS at 1-31 to 1-33; *compare* 2016 ROD at 14-16, 23-24 *with* 2021 ROD at 14-15, 23-24.

noncancer health hazards.[60] EPA considered whether to focus on additional COCs in OU4 but correctly found that over 98% of the total cancer and noncancer human health incremental risk in OU4 was posed by the same COCs identified for OU2.[61]

Likewise, the ecological risks that exist in OU2 are substantially similar to the risks EPA identified in OU4.[62] Additionally, despite some differences in risk posed to the benthic invertebrate community between OU2 and OU4, the relative risk conclusions for OU2 and OU4 are indeed substantially similar when averaged across all ecological receptors (i.e., benthic invertebrates, fish, crab, birds, and mammals).[63]

Given the distribution of contaminated sediments throughout OU4 (including OU2), the similarity of COCs, and the substantially similar human health and ecological risks, it is entirely appropriate, fair, and reasonable for EPA to consider the allocation in determining the Settling Parties' share of response costs in OU4.

III. OxyChem is Primarily Responsible for the Passaic River Remediation Because it Contributed Almost All of the TCDD in the River and Added to All Other COCs.

It is well documented, with information available to EPA and the Allocator, that OxyChem contributed almost all of the TCDD in the River and also contributed all other COCs. For decades, OxyChem's corporate predecessor, Diamond Shamrock Chemicals Company ("Diamond"), and Diamond's predecessors knowingly, intentionally, and illegally discharged significant amounts of contaminated waste—including dioxins—from the chemical manufacturing facility located at the Lister Facility into the River.[64] In 1992, the New Jersey Superior Court found "overwhelming evidence . . . that Diamond knew about the release of dioxins from its plant and the migration of these substances to surrounding areas" and that "[t]he only conclusion to be drawn is that Diamond's management was wholly indifferent to the consequences flowing from its decision.

[60] 2016 ROD at 22-23; 2021 ROD at 25 ("The BHHRA identified TCDD TEQ and PCBs as COCs, primarily due to consumption of LPRSA fish and crabs."); *see also* 2021 ROD at App. II, Tables 7-2a, 7-2b.

[61] Lower 17-Mile RI Report at 162-74; Exh. C at 3-2.

[62] *See* OU2 FFS at 1-32 to 1-33; Lower 17-Mile RI Report at 175-956; 2021 ROD at 35-36; Exh. B at 13-15.

[63] Exh. B at 13-15.

[64] *Aetna II*, 609 A.2d at 446-49, 454-55, 461-64 (noting that, "of the [federal] government's stockpile of Agent Orange, the average dioxin content of the product manufactured by Diamond was greater than that of the product manufactured by the four other companies whose products were stored [in Mississippi]"); *Diamond Shamrock Chems. Co. v. Aetna Cas. & Sur. Co.*, No. C-3939-84, at 8-12 (N.J. Ch. Apr. 12, 1989) ("*Aetna I*"); *see* Allocation Facility Data Reports (OxyChem) at 20-30, Att. J to Allocation Report.

Profits came first."[65] As the established legal successor to Diamond, OxyChem is responsible for the dioxin contamination in the Passaic River.[66]

There is broad scientific consensus that the Lister Facility is the primary source of TCDD in the River.[67] The specific dioxin contamination from the Lister Facility has been identified throughout the surficial zone.[68] One study estimates that the Lister Facility is responsible for 94% of the TCDD contamination in the River.[69]

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[65] *Aetna II*, 609 A.2d at 462-63.

[66] *See* Spill Act Litigation, Consent Judgment ¶ 21.14, *supra* note 17; Spill Act Litigation, Order Partially Granting Plaintiff's Motion for Partial Summary Judgment (N.J. Super. Ct. Law Div. July 19, 2011), ECF No. 951-29 in OxyChem's Litigation (concluding that OxyChem is the undisputed legal successor by merger to Diamond and that OxyChem is responsible for the liabilities of the original Diamond Shamrock Corporation, ultimately leading to settlement); Letter Order (Sept. 11, 2020), ECF No. 1105 at 4-5 in OxyChem's Litigation (concluding that OxyChem is the successor to Diamond); *see also Smith Land & Improvement Corp. v. Celotex Corp.*, 851 F.2d 86, 92 (3d Cir. 1988) ("Congress intended to impose successor liability on corporations which either have merged with or have consolidated with a corporation that is a responsible party as defined in [CERCLA]."); *Gould, Inc. v. A & M Battery & Tire Serv.*, 987 F. Supp. 353, 372 (M.D. Pa. 1997), *rev'd on other grounds*, 232 F.3d 162 (3d Cir. 2000) (concluding that a predecessor and a successor are indistinguishable in equity when assessing corporate liabilities); *Responsible Env't Solutions All. v. Waste Mgmt., Inc.*, No. 3:04cv013, 2011 WL 382617, at *1, 10 (S.D. Ohio Feb. 3, 2011) (agreeing that a successor's lack of participation in its predecessor's bad acts is "of no matter" and does not affect the successor's responsibility for response costs because "the successor stands in the shoes of its predecessors"). Because it has been judicially established that OxyChem is legally responsible for the operations of the Lister Facility, this Comment refers to Diamond and OxyChem interchangeably in Part III.

[67] *See* James D. Quadrini et al., *Fingerprinting 2,3,7,8-Tetrachlorodibenzodioxin Contamination Within the Lower Passaic River*, 34 ENV'T TOXICOLOGY & CHEM. 1485, 1485, 1496 (2015); Thomas J. Belton et. al., *A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Select Finfish, Crustaceans and Sediments of New Jersey Waterways* 4, 10, 13, 22 (N.J. Dep't of Env't Prot. Off. of Sci. & Rsch. Oct. 30, 1985); Bopp, *supra* note 3, at 955 (noting that TCDD remains the dominant dioxin contaminant in the River); Zongwei Cai et al., *Levels of Polychlorodibenzo-p-dioxins and Dibenzofurans in Crab Tissues from the Newark/Raritan Bay System*, 28 ENV'T SCI. & TECH. 1528, 1528, 1532 (1994); Zongwei Cai et al., *Response to Comments on "Levels of Polychlorodibenzo-p-dioxins and Dibenzofurans in Crab Tissues from the Newark/Raritan Bay System."* 30 ENV'T SCI. & TECH. 723, 723-24 (1996); Peter H. Israelsson et al., May 1, 2014, *Fate and Transport of Hydrophobic Organic Chemicals in the Lower Passaic River: Insights from 2,3,7,8-Tetrachlorodibenzo-p-Dioxin*, Estuaries & Coasts (2013); Mohammed Khairy et al., *Changing Sources of Polychlorinated Dibenzo-p-Dioxins and Furans in Sediments and Ecological Risk for Nekton in the Lower Passaic River and Newark Bay, New Jersey, USA*, 35 ENV'T TOXICOLOGY & CHEM. 550, 550, 560-61 (2016); Robert Parette et al., *Reconstruction of Historical 2,3,7,8-Tetrachlorodibenzo-p-dioxin Discharges from a Former Pesticide Manufacturing Plant to the Lower Passaic River*, 212 CHEMOSPHERE 1125, 1126, 1131 (2018).

[68] Khairy, *supra* note 67, at 557, 560.

[69] *Id.* at 558; *see also Aetna II*, 609 A.2d at 462 (describing the release of dioxins from the Lister Facility as "continuous").

In addition to TCDD, grossly inadequate waste disposal and housekeeping practices at the Lister Facility resulted in the contribution of other COCs to the River.[70] Between approximately 1946 and 1957, the Lister Facility manufactured as much as 100 million pounds of DDT.[71] Discharges of DDT from the Lister Facility were so substantial that they resulted in a "mountain" of solid DDT forming in the River, which Lister Facility employees would physically break up under the cover of darkness to avoid detection by the Coast Guard.[72] The Lister Facility also produced, used, and generated a range of other chemicals in connection with the manufacture of pesticides, including dieldrin,[73] PCBs,[74] and PAHs.[75] Elevated concentrations of all three of these COCs have been detected in River sediments collected immediately adjacent to the Lister Facility and are attributable to its operations.[76] Earlier sampling and investigations found all eight COCs to be present in Lister Facility soils and groundwater.[77]

Even by the standards that existed during the time of the Lister Facility's operation, waste was not disposed of in an appropriate or legal manner.[78] In the 1940s and 1950s, the Lister Facility

[70] *Aetna I*, No. C-3939-84, at 9-11.

[71] *Aetna II*, 609 A.2d at 447-48; Diamond Shamrock Chems. Co., *Site Evaluation 80 Lister Avenue Volume 1*, submitted to N.J. Dep't of Env't Prot., Table 2.2.1-2 (Feb. 1985), www.nj.gov/dep/passaicdocs/docs/upland/71.pdf ("Diamond Site Evaluation").

[72] *See Aetna II*, 609 A.2d at 448.

[73] The Lister Facility used dieldrin as an additive to DDT and in the manufacture of "Black Leaf" products. *See* Marion N. Gleason et al., CLINICAL TOXICOLOGY OF COMMERCIAL PRODUCTS 194, 734 (3d ed. 1969); Diamond Black Leaf Co., Advertisement No. 7431 pub. in PAC. COAST NURSERYMAN (1955); Donald E. H. Frear, ed., PESTICIDE HANDBOOK 36 (8th ed. 1956).

[74] Because several chlorobenzenes with high potential for PCB contamination (e.g., HCB, MCB, DCB, T3CB, and T4CB) were produced or used at the Lister Facility, those operations also likely generated PCBs as byproducts. ChemRisk, *Potential Sources of Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans in the Newark Bay Watershed*, Second Progress Report Table 2-7 (Nov. 30, 1990); Diamond Site Evaluation at Table 2.2.1-1.

[75] The Lister Facility produced PAHs, in particular naphthalene and 2-methylnapthalene, which are associated with a-naphthaleneacetic acid and DDT production that occurred at the Lister Facility. *See* R.H. Wellman, *Synthetic Chemicals for Agriculture Part 2: Fungicides, Nematocides, Rodenticides, and Weed Killers*, CHEM. INDUS. 223, 223-29 (Aug. 1948); Yoshiro Ogata et al., *Preparation of a-Naphthaleneacetic Acid by the Condensation of Napthalene with Chloroacetic Acid*, 72 J. OF THE AM. CHEM. SOC'Y 4302 (1949); Diamond Site Evaluation at Table 2.2.1-2.

[76] Allocation Facility Data Reports (OxyChem) at 23-25, 30, 44, 47, 54; Request for Authorization App. C-1 at 3-4.

[77] *See* NUS Corp., *Priority Pollutant Analytical Results from 80 Lister Avenue Study Area, Newark, New Jersey*, prepared for E.P.A., at 1, 12-16 (Mar. 30, 1984), www.nj.gov/dep/passaicdocs/docs/upland/55.pdf.

[78] *See Aetna I*, No. C-3939-84, at 11 (stating that, even by the standard of the 1950s and 1960s, OxyChem's conduct was "unacceptably wrong and irresponsible"); *Aetna II*, 609 A.2d at 463 (explaining that, although OxyChem's conduct should not be judged from "the vantage of twenty-twenty hindsight," the court "cannot ignore reality by accepting the blithe assurance of Diamond that it did not intend to injure others" because "[t]he evidence abounds the other way").

was subject to inspections by the New York Harbor District, which was "concerned with keeping oil and acids out of the water because of hinderances to navigation," and periodic inspections by the PVSC.[79] Although the PVSC requested by 1956 that Lister Facility send its discharges to the sanitary sewer and made clear that pollution of the River was illegal, the Lister Facility continued to discharge pollutants directly to the River.[80]

OxyChem was well aware of the toxicity of the Lister Facility waste, including dioxins.[81] It was also well aware of discharge laws and options to comply with them.[82] Nevertheless, it knowingly violated laws and regulations by discharging waste to the River while actively attempting to avoid detection by authorities.[83] Notably, OxyChem rerouted lines containing hydrochloric acid to discharge "underneath the surface of the water [of the River] so that they were never detected as a source of acid effluent."[84] It also employed a secret alarm system to alert the foreman when an inspector was present so employees could conceal the discharges to the River.[85] Had OxyChem merely complied with existing laws, there would be dramatically less dioxin in the River.

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[79] Memo. From John Burton to B. D. Gleisner Re: Newark Effluent Problem (July 10, 1956) ("Burton 1956 Memo").

[80] *See* Allocation Facility Data Reports (OxyChem) at 31; *Aetna II*, 609 A.2d at 462-64 (recounting testimony from employees about Lister Facility practices and describing OxyChem's "heedless indifference to the environmental damage which resulted from its manufacturing operations"). For example, OxyChem discharged waste from the cleaning of equipment and spills to the River through trenches leading from building floors, *Aetna I*, No. C-3939-84, at 9; *Aetna II*, 609 A.2d at 463; failed to contain solid waste in drums and landfill it, instead stockpiling the drums near the River and, later, dumping the waste directly into the River, Arthur Scureman Test. at 38-40 (Oct. 17, 1988) in *Aetna I*; *see Aetna II*, 609 A.2d at 447-48; allowed untreated, contaminated surface water to run off from the Facility into the River both due to the general topography of the property and through a system of 16- to 24-inch diameter trenches, pipes, and drains that flowed into to the River, Diamond Site Evaluation at ¶¶ 2.2.1, 3.1.2; *Aetna II*, 609 A.2d at 450; *Aetna I*, No. C-3939-84, at 9; carelessly handled waste such that it was left exposed to the elements on the ground outdoors and would enter the River during storm and flood events, Federal Emergency Management Agency, Flood Insurance Rate Map, Essex County, New Jersey, Map No. 34013C0157F (June 4, 2007); *see Aetna I*, No. C-3939-84, at 10, 32; *Aetna II*, 609 A.2d at 448-49, 463. This was one way in which OxyChem specifically contributed to the DDT contamination of the River. The Lister Facility handled large volumes of DDT outdoors and had no incentive to handle it with care. John Burton Test. at 40-41 (Oct. 17, 1988) in *Aetna I*.

[81] *Aetna I*, No. C-3939-84, at 33 ("But Diamond did know the nature of the chemicals it was handling, it did know that they were being continuously discharged into the environment, and it did know that they were doing at least some harm"); *Aetna II*, 609 A.2d at 447.

[82] *See Aetna I*, No. C-3939-84, at 8-9 ("Diamond was conscious that its discharges into the river were illegal. It deliberately concealed them . . . ."); *Id.* at 33 ("Diamond unequivocally knew that at least some of this contaminating activity violated the then existing statutory prohibitions against discharges into the Passaic River.").

[83] *Id.* at 8-9; *Aetna II*, 609 A.2d at 455 (describing OxyChem's actions as a "deliberate and willful course of misconduct").

[84] Allocation Facility Data Reports (OxyChem) at 27; Burton 1956 Memo.

[85] *Aetna I*, No. C-3939-84, at 9.

Similarly, OxyChem affirmatively chose not to implement best practices for treating and disposing of the Lister Facility waste, despite knowing that they could have done so. For example, at that time, the best practice for disposal of its process waste would have been to treat it and discharge it to the PVSC.[86] Although it was aware of this option, OxyChem elected not to comply due to cost concerns.[87] Even after receiving citations for its illegal discharges, OxyChem only feigned compliance, spending just enough money to mislead authorities by diverting a fraction of its process waste to the PVSC while it discharged the remainder to the River illegally.[88]

Even after the Lister Facility ceased operation, OxyChem continued to avoid responsibility for the amount and toxicity of contaminants it discharged to the River. Rather than accepting the scientific community's overwhelming consensus on the toxicity of and risk from TCDD, OxyChem and its indemnitors have spent decades seeding the literature with scientifically unsound studies designed to thwart the needed and costly cleanup of the River.[89] They funded hundreds of studies over the years in an attempt to downplay the risk from TCDD in the River.[90]

The Allocation Report properly concluded that OxyChem is primarily responsible for the Site remediation even though several aspects of the Allocation methodology and calculations benefitted OxyChem relative to other parties. For example, culpability (or bad acts) and cooperation, though commonly employed as stand-alone factors in allocations, here were used only as adjustment factors (so they cannot independently lead to a significant share of responsibility), which discounts OxyChem's egregious conduct that led to the extensive TCDD pollution in the River.[91] Contributions from the direct discharge pathway, which are well documented from OxyChem's facility, were assumed for other parties' facilities that operated before 1924 (when the PVSC began operation) using large waste volume estimates.[92] Furthermore, overland fate and transport ("OFT") pathway contributions, which are inherently more difficult to calculate than direct discharges (and that EPA had previously stated were not a

[86] *See* Allocation Facility Data Reports (OxyChem) at 31; *see* May 2020 Koch Report § 6.3, Att. O to Allocation Report.

[87] *Aetna I*, No. C-3939-84, at 9-10; *Aetna II*, 609 A.2d at 462 ("Profits came first.").

[88] *Aetna I*, No. C-3939-84, at 9; *Aetna II*, 609 A.2d at 448; Allocation Facility Data Reports (OxyChem) at 31, 39-40; Memo. from J. Burton to H. S. Weiner (Apr. 4, 1960); PVSC Notice of Violation to Diamond Alkali Co. (Aug. 3, 1956).

[89] Exh. A at 8.

[90] *Id.*

[91] Allocation Report at 32-34; *see Env't Transp. Sys., Inc. v. ENSCO, Inc.*, 969 F.2d 503, 509, 512 (7th Cir. 1992) (stating that, "in any given case, a court may consider several factors, a few factors, or only one determining factor, . . . depending on the totality of circumstances presented to the court" and upholding the allocation of all costs to one party based on fault).

[92] *See* Allocation Report at 20-23; Allocation Facility Data Reports (OxyChem) at 12, 25, 31, 36, 39.

significant pathway of COCs to the River), were included.[93] These contributions were calculated by using a maximum COC concentration in soil on a sitewide basis (regardless of location) and by using property size to determine the area subject to erosion, which, along with the application of a conservative annual soil erosion rate, resulted in increased relative contributions from certain non-OxyChem parties, such as Nokia and Pharmacia.[94] This conservative approach to addressing data gaps also did not account for facility-specific factors such as topography, distance from the River, impediments to sheet flow to the River, and stormwater management features.[95] The fact that these allocation features or calculations served to lower OxyChem's share lends further confidence to the conclusion that this is fundamentally a one-party site.[96]

IV. The Settlement Includes a Significant Premium That Mitigates the Impact of Potential Challenges to Specific Allocation Calculations or Party Findings.

If the United States moves for entry of the CD in the District Court, the Court will evaluate the CD according to three criteria: (1) fairness—both substantive and procedural, (2) reasonableness, and (3) consistency with CERCLA's goals.[97] This settlement meets each of these criteria. It was entered into after an arm's length negotiation that complied with all procedural requirements, it is based on a reasonable foundation that includes the guidance of the Allocation Report, and it is for an amount that far exceeds the Settling Parties' actual responsibility at the Site.

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[93] *See* Allocation Report at 19, 21; Allocation Protocol at 6; 2016 ROD at 18-19.

[94] Allocation Report at 21-22; Allocation Facility Data Reports (Nokia-Lucent Technologies Inc., Pharmacia). As discussed, most of OxyChem's releases of all eight COCs, including its contribution of substantially all of the dioxin in the River, consisted of direct (and illegal) discharges. Allocation Facility Data Reports (OxyChem) at 3-40. In contrast, very few non-OxyChem Allocation Parties' COC contributions were through direct pathways. *See generally* Allocation Facility Data Reports. The overwhelming majority of COCs in OU2 sediments attributed by the Allocator to non-OxyChem Allocation Parties were through OFT, which often occurs by unintentional conduct and is difficult to calculate accurately given the number of assumptions that must be made. *Id.*

[95] *See* Allocation Report at 21-22. For example, Pharmacia's highest concentration of PCBs was 560 feet from the Passaic River, and there was crushed stone, buildings, and a vegetative berm between this soil and the River, making it improbable that such concentrations actually reached the River. *See* Allocation Facility Data Reports (Pharmacia) at 4, 15. Pharmacia's extensive soil sampling data showed sharply declining concentrations from the point of the maximum concentration and numerous non-detect results, including near the River. Allocation Facility Data Reports (Pharmacia) at 15, 18. Similarly, Nokia's highest PCB soil sample was well away from the River, which does not suggest that it was a source of discharge to the River. Allocation Facility Data Reports (Nokia-Lucent Technologies Inc.) at 18-19. Notwithstanding, Nokia and Pharmacia support the settlement.

[96] Notably, to resolve the State of New Jersey's related Spill Act Litigation, the parties affiliated with the Lister Facility, including OxyChem, were required to pay roughly 90% of the State's total settlement amounts. *See* Spill Act Litigation, Consent Judgment, *supra* note 17.

[97] *In re Tutu Water Wells CERCLA Litig.*, 326 F.3d 201, 207 (3d Cir. 2003); *see* 42 U.S.C.A. § 9613(j)(2) (stating that the District Court must determine if EPA's decision to execute a proposed CD is "arbitrary and capricious or otherwise not in accordance with law").

Germane to the analysis of all three criteria is the amount of the settlement premium applied. Cashout settlements like the CD traditionally involve the application of a premium based on the risk of additional later costs that the United States will be precluded from recovering from the Settling Parties.[98]

Here, the Allocator determined that the Settling Parties were responsible for a combined share of only 0.0166217% of EPA's estimated $1.82 billion in future cleanup costs for OU2 and OU4 of the Site.[99] Strict adherence to the allocation would set the Settling Parties' collective responsibility at only $302,514.[100] The Settling Parties' settlement payment of $150 million is facially fair and reasonable because it is *hundreds* of times larger than the Settling Parties' allocated collective responsibility.[101]

The premium doubles if the Settling Parties' past costs are considered. In addition to the $150 million that would be due under the proposed settlement, the Settling Parties previously paid more than $160 million in response costs at the Site. The vast majority of these costs were incurred under prior settlement agreements with the United States: $10.78 million under the 2004 Agreement to fund EPA's work on the Remedial Investigation/Feasibility Study ("RI/FS") for the River;[102] $111.69 million under the 2007 Administrative Settlement Agreement for completion of the Remedial Investigation/Feasibility Study;[103] and $23.45 million under the River Mile 10.9 Removal Administrative Settlement Agreement.[104] The Settling Parties have also incurred another approximately $10.12 million in costs they could seek from OxyChem tied to the cleanup of OU2 and OU4, including but not limited to legal fees, administrative costs, costs of investigation, planning, and technical support, and other indirect costs incurred for work that has significantly

[98] *See United States v. Rohm & Haas Co.*, 721 F. Supp. 666, 673-74 (D.N.J. 1989) (describing a cashout settlement premium as the settling parties' payment of "additional funds above their volumetric share of the waste in exchange for avoiding the possible joint and several liability and substantial litigation costs which accompany being a CERCLA defendant").

[99] *See* Notice of Lodging, 87 Fed. Reg. 78710; Allocation Operations Spreadsheet.

[100] Non-OxyChem Allocation Parties' collective minor responsibility would be only $1,099,092.

[101] *See United States v. IMC E. Corp.*, No. 18-CV-3818(GRB)(ARL), 2022 WL 4134321, at *6 (E.D.N.Y. Sept. 12, 2022) (concluding that a proposed CERCLA consent decree was reasonable based on the defendant's payment of "more than a 100% premium on its actual liability in exchange for an early settlement").

[102] Administrative Settlement Agreement, CERCLA Docket No. 02-2004-2011 (Apr. 6, 2004), semspub.epa.gov/src/document/02/99861.

[103] Administrative Settlement Agreement and Order on Consent for Remedial Investigation/Feasibility Study, CERCLA Docket No. 02-2007-2009 (May 8, 2007), semspub.epa.gov/src/document/02/99864.

[104] Administrative Settlement Agreement and Order on Consent for Removal Action, CERCLA Docket No. 02-2012-2015 (June 18, 2012), semspub.epa.gov/src/document/02/232657.

benefitted the entire cleanup effort.[105] Finally, the 2018 Settlement Agreement Parties and 2021 Settlement Agreement Parties, as those terms are defined in the CD,[106] previously paid $5,892,600, collectively, to settle their alleged CERCLA liability for OU2 of the Site.[107]

The Settling Parties have contribution claims under CERCLA against OxyChem and other non-settling PRPs arising from these past costs. However, under the proposed CD, the Settling Parties would waive these cost recovery claims (with limited exceptions), effectively releasing these claims and conferring a benefit on non-settling parties. Thus, the value of the settlement, combining new cash and released claims, is over $300 million, roughly one thousand times the Settling Parties' collective share of responsibility, guaranteeing that the settlement is facially fair and reasonable. Although allocations may be inherently imperfect,[108] the premium paid by the Settling Parties ensures that this settlement is fair, reasonable, and consistent with CERCLA's goals regardless of any potential imperfections in the Allocation.[109]

V. Conclusion

TCDD is the standard by which the toxicity of all other COCs is measured; EPA was right to focus its remedy on addressing the risks from TCDD in its remedy for OU2 and OU4. EPA should focus responsibility for Site costs on the party that contributed substantially all of the TCDD to the River: OxyChem. It is OxyChem that is responsible for the highest level of contamination of the River, and thus it is OxyChem that should bear the costs of—and perform the work for—its remediation.

Nevertheless, the Settling Parties to this proposed CD have agreed to an aggregate settlement to address their COC contributions to the River that *far* exceeds their actual responsibility, by any reasonable measure. The settlement was reached through a procedurally fair

[105] As part of the CD, the Settling Parties agreed to waive certain contribution claims they would have against other PRPs. *See, e.g.*, CD ¶ 20.

[106] *See* CD at 5.

[107] *See* Administrative Settlement Agreement, CERCLA Docket No. 02-2017-2023 (Nov. 14, 2017), semspub.epa.gov/src/document/02/518131; Administrative Settlement Agreement, CERLCA Docket No. 02-2020-2013 (Apr. 22, 2020), semspub.epa.gov/src/document/02/591178.

[108] *See United States v. Kramer*, 19 F. Supp. 2d 273, 282 (D.N.J. 1998); *Rohm & Haas Co.*, 721 F. Supp. at 685-86.

[109] *See Kramer*, 19 F. Supp. 2d at 288 (noting, in approving a consent decree, that the "'ideal' share of liability—based on perfect knowledge of harm caused by these parties only, expressed as a proportion of the total costs of remediation at the site—is undoubtedly less than the compromise figure reached for this group" and that "[t]hese parties are paying a disproportionate share of their liability measured in 'ideal' terms" (alteration, citation, and quotation marks omitted)).

negotiation process, it is supported by sound data, and it is a patently reasonable agreement with sizeable benefits to the public, as is consistent with CERCLA's purposes.[110]

For the reasons stated above, Commenters urge the United States to support the CD as executed and to move for entry of the CD in the District Court. The Commenters also urge the United States to promptly engage in good faith settlement negotiations with the remaining participating allocation parties (whose shares are also minor) as well as the remaining defendants and third-party defendants in OxyChem's Litigation.

Thank you for your consideration.

Respectfully submitted,

/s/ *Jeffrey D. Talbert*
PRETI, FLAHERTY, BELIVEAU & PACHIOS, LLP
PO Box 9546
One City Center
Portland, ME 04112
Tel: 207.791.3239
Jeffrey D. Talbert, Esq.
Liaison Counsel for the Small Parties Group

/s/ *John F. Gullace*
MANKO, GOLD, KATCHER & FOX, LLP
Three Bala Plaza East, Suite 700
Bala Cynwyd, PA 19004
Tel: 484.430.5700
John F. Gullace, Esq.
Counsel for Pharmacia LLC

/s/ *Michael D. Lichtenstein*
LOWENSTEIN SANDLER LLP
One Lowenstein Drive
Roseland, NJ 07068
Tel: 973.597.2408
Michael D. Lichtenstein, Esq.
Counsel for Nokia of America Corp.

[110] *See* 42 U.S.C.A. § 9622(a) ("Whenever practicable and in the public interest, as determined by the President, the President shall act to facilitate agreements under this section that are in the public interest and consistent with the National Contingency Plan in order to expedite effective remedial actions and minimize litigation.").

Enclosures:

Table of Authorities Listing All Sources Cited
Appendix 1 – SPG Parties Commenting
Exhibit A – Linda S. Birnbaum, *Report* (Mar. 21, 2023)
Exhibit B – D. Michael Johns, *Relative Risks of OU2 Contaminants of Concern & Comparison with OU4: A Summary of Data Information Regarding Relative Ecological Risks for the Lower Passaic River* (Mar. 22, 2023)
Exhibit C – Betsy Ruffle, *Relative Risks of OU2 Contaminants of Concern & Comparison with OU4: A Summary of Data and Information Regarding Relative Human Health Risks for the Lower Passaic River* (Mar. 22, 2023)

RE: Table of Authorities Cited in Small Parties Group Joint Comments on Proposed CERCLA Consent Decree Regarding the Diamond Alkali Superfund Site: *United States of America v. Alden Leeds, Inc., et al.*, Civil Action No. 2:22-cv-07326 (MCA-LDW) (D.N.J.); D.J. Ref. No. 90-11-3-07683/1

Note: All PAP docs accessible at:
cumulis.epa.gov/supercpad/SiteProfiles/index.cfm?fuseaction=second.scs&id=0200613&doc=Y&colid=41378&region=02&type=SC

*post-dates allocation and/or settlement

	Authority	Location
Statutes		
1	42 U.S.C.A. § 9602(a)	uscode.house.gov
2	42 U.S.C.A. § 9613(j)(2)	uscode.house.gov
3	42 U.S.C.A. § 9621(d)(1)	uscode.house.gov
4	42 U.S.C.A. § 9622(a)	uscode.house.gov
Allocation Documents		
5	AlterEcho, Final Allocation Recommendation Report (Dec. 28, 2020) ("Allocation Report")	semspub.epa.gov/src/document/02/609904
6	Letter from Eric J. Wilson, EPA, to OU2 PRPs (Sept. 18, 2017)	Att. A to Allocation Report
7	AlterEcho, Diamond Alkali Superfund Site OU2 Allocation Guide (June 15, 2018, as amended, July 6, 2018, and Mar. 22, 2019) ("Allocation Guide")	Att. G to Allocation Report
8	AlterEcho, Diamond Alkali Superfund Site OU2 Allocation Protocol ("Allocation Protocol"	Att. H to Allocation Report
9	List of Participating Allocation Parties	Att. I to Allocation Report
10	Allocation Facility Data Reports	Att. J to Allocation Report
11	Allocation Operations Spreadsheet	Att. K to Allocation Report
12	May 2020 Koch Report	Att. O to Allocation Report
Federal Register		
13	*Notice of Lodging of Proposed Consent Decree Under the Comprehensive Environmental Response, Compensation, and Liability Act, 87 Fed. Reg. 78710 (Dec. 22, 2022), as amended by 88 Fed. Reg. 2133 (Jan. 12, 2023) ("Notice of Lodging")	www.federalregister.gov
14	Toxic and Hazardous Substances Control - Contractor and Subcontractor Access to Confidential Business Information, 52 Fed. Reg. 19921 (May 28, 1987)	www.federalregister.gov
15	Notice of Request for Public Comment, 50 Fed. Reg. 5034 (Feb. 5, 1985)	www.federalregister.gov

	Authority	Location
16	Amendment to National Oil and Hazardous Substance Contingency Plan, National Priorities List, 49 Fed. Reg. 37070 (Sept. 21, 1984) (codified at 40 C.F.R. Part 300) ("National Priorities List")	www.federalregister.gov
Regulatory Documents		
17	AECOM, *Baseline Human Health Risk Assessment for the Lower Passaic River Study Area* (July 2017)	semspub.epa.gov/work/02/616138.pdf
18	*Anchor QEA, LLC, *Remedial Investigation Report: Lower Passaic River Study Area Remedial Investigation/Feasibility* (July 2019) ("Lower 17-Mile RI Report")	ourpassaic.org/RemedialInvestigation.aspx
19	Human Health Risk Assessment	App. D of the OU2 FFS
20	*Integral Consulting Inc., *Upper 9-Mile Source Control Interim Remedy Feasibility Study, Lower Passaic River Study Area Remedial Investigation and Feasibility Study* (Sept. 9, 2021) ("Upper 9-Mile IR FS")	semspub.epa.gov/work/02/625207.pdf
21	Letter from Eric J. Wilson, EPA, to Marcia E. Backus, Occidental Petroleum Corp. (Nov. 28, 2017)	ECF No. 2246-6 in OxyChem's Litigation
22	Letter from Nicoletta Di Forte, EPA, to PRPs (Mar. 31, 2016)	PAP-00204570
23	The Louis Berger Group et al., *Lower Eight Miles of the Lower Passaic River: Remedial Investigation Report for Focused Feasibility Study* (Mar. 3, 2014) ("OU2 FFS")	semspub.epa.gov/src/document/02/703639
24	U.S. E.P.A., Memo. Re: Request for Authorization to Conduct a CERCLA Non-Time-Critical Removal Action at the Diamond Alkali Site, Newark, Essex County, New Jersey (Jan. 8, 2009) ("Request for Authorization")	semspub.epa.gov/src/document/02/239617
25	U.S. E.P.A., Record of Decision: Lower 8.3 Miles of the Lower Passaic River Part of the Diamond Alkali Superfund Site (Mar. 3, 2016) ("2016 ROD")	semspub.epa.gov/src/document/02/396055
26	*U.S. E.P.A., Record of Decision for an Interim Remedy in the Upper 9 Miles of the Lower Passaic River Study Area, OU4 of the Diamond Alkali Superfund Site (Sept. 28, 2021) ("2021 ROD")	semspub.epa.gov/src/collection/02/AR65669
27	Windward Env't LLC, *Lower Passaic River Study Area Baseline Ecological Risk Assessment* (June 17, 2019)	semspub.epa.gov/work/02/620400.pdf
Prior Agreements		
28	Administrative Settlement Agreement, CERCLA Docket No. 02-2004-2011 (Apr. 6, 2004)	semspub.epa.gov/src/document/02/99861
29	Administrative Settlement Agreement and Order on Consent for Remedial Investigation/Feasibility Study, CERCLA Docket No. 02-2007-2009 (May 8, 2007)	semspub.epa.gov/src/document/02/99864
30	Administrative Settlement Agreement and Order on Consent for Removal Action, CERCLA Docket No. 02-2012-2015 (June 18, 2012)	semspub.epa.gov/src/document/02/232657
31	Administrative Settlement Agreement, CERCLA Docket No. 02-2017-2023 (Nov. 14, 2017)	semspub.epa.gov/src/document/02/518131

	Authority	Location
32	Administrative Settlement Agreement, CERLCA Docket No. 02-2020-2013 (Apr. 22, 2020)	semspub.epa.gov/src/document/02/591178
Government Documents		
33	Federal Emergency Management Agency, Flood Insurance Rate Map, Essex County, New Jersey, Map No. 34013C0157F (June 4, 2007)	map1.msc.fema.gov/firm?id=34013C0157F
34	N.J. Dep't of Env't Prot., Memo Re: Lower Passaic River - Phase I Removal Action Surface Water Discharge Permit Equivalent (Dec. 10, 2010) ("NJDEP Memo")	
35	N.J. Exec. Order No. 40 (June 2, 1983)	PAP-00143133
36	U.S. E.P.A., *Learn About Dioxin*	www.epa.gov/dioxin/learn-about-dioxin (last visited Mar. 15, 2023)
37	U.S. E.P.A. Press Release, *Occidental Chemical Signs Consent Order for Storage and Destruction of Love Canal Wastes* (June 1, 1989)	epa.gov/archive/epa/aboutepa/occidental-chemical-signs-consent-order-storage-and-destruction-love-canal-wastes.html
38	U.S. E.P.A., Memo Re: Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions, Off. of Solid Waste & Emergency Response Directive 9355.0-30 (Apr. 22, 1991)	nepis.epa.gov/exe/zypurl.cgi?dockey=910165cr.txt
39	*Yeh Decl.	ECF No. 84-1 in *Alden Leeds*
Technical Publications		
40	Thomas J. Belton et. al., *A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Select Finfish, Crustaceans and Sediments of New Jersey Waterways* 4 (N.J. Dep't of Env't Prot. Off. of Sci. & Rsch. Oct. 30, 1985)	https://www.state.nj.us/dep/dsr/dioxin/Study%20of%20Dioxin.pdf
41	Richard F. Bopp et al., *A Major Incident of Dioxin Contamination: Sediments of New Jersey Estuaries*, 25 ENV'T. SCI. & TECH. 951 (1991)	PAP-00162270
42	Zongwei Cai et al., *Levels of Polychlorodibenzo-p-dioxins and Dibenzofurans in Crab Tissues from the Newark/Raritan Bay System*, 28 ENV'T SCI. & TECH. 1528 (1994)	http://www.water.rutgers.edu/Projects/EPA_Raritan_River_Project/08_Data/Papers&Articles/Cai%20et%20al%201994.pdf
43	Zongwei Cai et al., *Response to Comments on "Levels of Polychlorodibenzo-p-dioxins and Dibenzofurans in Crab Tissues from the Newark/Raritan Bay System."* 30 ENV'T SCI. & TECH. 723 (1996)	https://pubs.acs.org/doi/pdf/10.1021/es9510065
44	ChemRisk, *Potential Sources of Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans in the Newark Bay Watershed*, Second Progress Report (Nov. 30, 1990)	PAP-00184772
45	Donald E. H. Frear, ed., PESTICIDE HANDBOOK (8th ed. 1956)	PAP-00397938
46	Marion N. Gleason et al., CLINICAL TOXICOLOGY OF COMMERCIAL PRODUCTS (3d ed. 1969)	PAP-00397979
47	Peter H. Israelsson et al., May 1, 2014, *Fate and Transport of Hydrophobic Organic Chemicals in the*	PAP-00149615

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	Lower Passaic River: Insights from 2,3,7,8-Tetrachlorodibenzo-p-Dioxin, Estuaries & Coasts (2013)	
48	Mohammed Khairy et al., *Changing Sources of Polychlorinated Dibenzo-p-Dioxins and Furans in Sediments and Ecological Risk for Nekton in the Lower Passaic River and Newark Bay, New Jersey, USA*, 35 ENV'T TOXICOLOGY & CHEM. 550 (2016)	PAP-00162231
49	Travis R. Kline, *Redefining Environmental Cost Allocation Using the Toxicity Factor*	alterecho.com/artfiledownload/8/CostAllocationTravisKline.pdf (last visited Mar. 17, 2023)
50	NUS Corp., *Priority Pollutant Analytical Results from 80 Lister Avenue Study Area, Newark, New Jersey*, prepared for E.P.A. (Mar. 30, 1984)	www.nj.gov/dep/passaicdocs/docs/upland/55.pdf
51	Yoshiro Ogata et al., *Preparation of a-Naphthaleneacetic Acid by the Condensation of Napthalene with Chloroacetic Acid*, 72 J. OF THE AM. CHEM. SOC'Y 4302 (1949)	PAP-00400430
52	Robert Parette et al., *Reconstruction of Historical 2,3,7,8-Tetrachlorodibenzo-p-dioxin Discharges from a Former Pesticide Manufacturing Plant to the Lower Passaic River*, 212 CHEMOSPHERE 1125 (2018)	PAP-00210169
53	James D. Quadrini et al., *Fingerprinting 2,3,7,8-Tetrachlorodibenzodioxin Contamination Within the Lower Passaic River*, 34 ENV'T TOXICOLOGY & CHEM. 1485 (2015)	PAP-00162743
54	R.H. Wellman, *Synthetic Chemicals for Agriculture Part 2: Fungicides, Nematocides, Rodenticides, and Weed Killers*, CHEM. INDUS. 223 (Aug. 1948)	PAP-00401714
Reports		
55	*Linda S. Birnbaum, *Report* (Mar. 21, 2023) (Exh. A)	Attached
56	* D. Michael Johns, *Relative Risks of OU2 Contaminants of Concern & Comparison with OU4: A Summary of Data Information Regarding Relative Ecological Risks for the Lower Passaic River* (Mar. 22, 2023) (Exh. B)	Attached
57	*Betsy Ruffle, *Relative Risks of OU2 Contaminants of Concern & Comparison with OU4: A Summary of Data and Information Regarding Relative Human Health Risks for the Lower Passaic River* (Mar. 22, 2023) (Exh. C)	Attached
Other Supporting Documents		
58	John Burton Test. (Oct. 17, 1988) in *Aetna I*	www.nj.gov/dep/passaicdocs/docs/SummaryJudgment/Exhibits/Exhibit19.pdf
59	Diamond Black Leaf Co., Advertisement No. 7431 pub. in PAC. COAST NURSERYMAN (1955)	PAP-00400429
60	Diamond Shamrock Chems. Co., *Site Evaluation 80 Lister Avenue Volume 1*, submitted to N.J. Dep't of Env't Prot. (Feb. 1985) ("Diamond Site Evaluation")	www.nj.gov/dep/passaicdocs/docs/upland/71.pdf

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61	Memo. From John Burton to B. D. Gleisner Re: Newark Effluent Problem (July 10, 1956) ("Burton 1956 Memo")	PAP-00143565
62	Memo. from J. Burton to H. S. Weiner (Apr. 4, 1960)	PAP-00155169
63	PVSC Notice of Violation to Diamond Alkali Co. (Aug. 3, 1956)	PAP-00143727
64	Arthur Scureman Test. (Oct. 17, 1988) in *Aetna I*	www.nj.gov/dep/passaicdocs/docs/SummaryJudgment/Exhibits/Exhibit18.pdf
Cases		
65	*Action Mfg. Co. v. Simon Wrecking Co.*, 428 F. Supp. 2d 288 (E.D. Pa. 2006), *aff'd*, 287 F. App'x 171 (3d Cir. 2008)	Westlaw
66	*AlliedSignal, Inc. v. Amcast Int'l Corp.*, 177 F. Supp. 2d 713 (S.D. Ohio 2001)	Westlaw
67	*Caldwell Trucking PRP Grp. v. Pullman Co.,* No. 95-1690 (DMC), 2002 U.S. Dist. LEXIS 28410 (D.N.J. Nov. 21, 2002), *aff'd sub nom. Caldwell Trucking PRP v. Rexon Tech. Corp.*, 421 F.3d 234 (3d Cir. 2005)	LEXIS
68	*Diamond Shamrock Chems. Co. v. Aetna Cas. & Sur. Co.*, No. C-3939-84 (N.J. Ch. Apr. 12, 1989) ("*Aetna I*")	www.nj.gov/dep/passaicdocs/docs/SummaryJudgment/Exhibits/Exhibit15.pdf
69	*Diamond Shamrock Chems. Co. v. Aetna Cas. & Sur. Co.*, 609 A.2d 440 (N.J. Super. App. Div. 1992) ("*Aetna II*")	Westlaw
70	*Env't Transp. Sys., Inc. v. ENSCO, Inc.*, 969 F.2d 503 (7th Cir. 1992)	Westlaw
71	*Gould, Inc. v. A & M Battery & Tire Serv.*, 987 F. Supp. 353 (M.D. Pa. 1997), *rev'd on other grounds*, 232 F.3d 162 (3d Cir. 2000)	Westlaw
72	*In re Tutu Water Wells CERCLA Litig.*, 326 F.3d 201 (3d Cir. 2003)	Westlaw
73	*Litgo N.J., Inc. v. Martin*, No. 06-2891 (AET), 2011 WL 65933 (D.N.J. Jan. 7, 2011)	Westlaw
74	*New York v. Solvent Chem. Co.*, 685 F. Supp. 2d 357 (W.D.N.Y. 2010), *vacated and remanded in part on other grounds*, 453 F. App'x 42 (2d Cir. 2011)	Westlaw
75	*N.J. Dep't of Env't Prot. v. Occidental Chem. Corp.*, No. ESX-L9868-05, Consent Judgment and Order on the Entry and Approval of the Consent Judgment (N.J. Super. Ct. Law Div. Dec. 16, 2014); Order Partially Granting Plaintiff's Motion for Partial Summary Judgment (July 19, 2011) ("Spill Act Litigation")	ECF Nos. 951-29, 951-30, 951-31 in OxyChem's Litigation
76	*Occidental Chem. Corp. v. 21st Century Fox Am., Inc.*, No. 2:18-cv-11273-MCA-LDW (D.N.J.) ("OxyChem's Litigation") *individual filings	PACER
77	*Responsible Env't Solutions All. v. Waste Mgmt., Inc.*, No. 3:04cv013, 2011 WL 382617 (S.D. Ohio Feb. 3, 2011)	Westlaw
78	*Smith Land & Improvement Corp. v. Celotex Corp.*, 851 F.2d 86 (3d Cir. 1988)	Westlaw

	Authority	Location
79	*Trinity Indus., Inc. v. Greenlease Holding Co.*, 903 F.3d 333 (3d Cir. 2018)	Westlaw
80	**United States v. Alden Leeds, Inc., et al.*, No. 2:22-cv-07326 (MCA-LDW) (D.N.J.) ("*Alden Leeds*")	PACER
81	*United States v. Cannons Eng'g Corp.*, 899 F.2d 79 (1st Cir. 1990)	Westlaw
82	*United States v. Hooker Chems. & Plastics Corp.*, 680 F. Supp. 546 (W.D.N.Y. 1988)	Westlaw
83	*United States v. Hooker Chems. & Plastics Corp.*, 722 F. Supp. 960 (W.D.N.Y. 1989)	Westlaw
84	**United States v. IMC E. Corp.*, No. 18-CV-3818(GRB)(ARL), 2022 WL 4134321 (E.D.N.Y. Sept. 12, 2022)	Westlaw
85	*United States v. Kramer*, 953 F. Supp. 592 (D.N.J. 1997)	Westlaw
86	*United States v. Kramer*, 19 F. Supp. 2d 273 (D.N.J. 1998)	Westlaw
87	*United States v. Rohm & Haas Co.*, 721 F. Supp. 666 (D.N.J. 1989)	Westlaw

Appendix 1 - SPG Parties Commenting

1. TFCF America, Inc. (f/k/a 21st Century Fox America, Inc.)
2. Akzo Nobel Coatings, Inc.
3. Arkema Inc.
4. Atlantic Richfield Company
5. BASF Corporation (on its own behalf and on behalf of BASF Catalysts LLC)
6. Bath Iron Works Corporation
7. Benjamin Moore & Co.
8. Canning Gumm LLC
9. Paramount Global (f/k/a ViacomCBS Inc.)
10. Chevron Environmental Management Company for itself and on behalf of Texaco Inc. and TRMI-H LLC
11. Clean Earth of North Jersey, Inc.
12. CNA Holdings LLC (by and through its indemnitor Essex County Improvement Authority)
13. Coats & Clark Inc.
14. CC Oldco Corporation (f/k/a Congoleum Corporation)
15. Conopco, Inc., d/b/a Unilever (as successor to CPC/Bestfoods, former parent of Penick Corporation)
16. Cooper Industries, LLC
17. Covanta Essex Company
18. Croda Inc.
19. DII Industries, LLC
20. EIDP, Inc. (f/k/a E. I. du Pont de Nemours and Company) on its own behalf and on behalf of Pitt-Consol Chemical Company
21. EnPro Industries, Inc., on behalf of itself and related corporate entities
22. EPEC Polymers, Inc., on behalf of itself and related corporate entities
23. Essex Chemical Corporation
24. Franklin-Burlington Plastics, Inc.
25. Garfield Molding Company, Inc.
26. General Electric Company
27. Goodrich Corporation for itself and for Kalama Specialty Chemicals, Inc., Noveon Kalama Inc. (f/k/a Kalama Chemical, Inc., f/k/a BFGoodrich Kalama, Inc.), and Emerald Kalama Chemical, LLC
28. L3Harris Technologies, Inc., successor in interest to Exelis Inc., successor in interest to the defense business of ITT Corporation
29. The Hartz Consumer Group, Inc. as successor to certain liabilities of The Hartz Mountain Corporation
30. Hexcel Corporation
31. Hoffmann-LaRoche Inc.
32. Honeywell International Inc.
33. Johnson & Johnson
34. Leemilt's Petroleum, Inc. (successor to Power Test of New Jersey, Inc.), on its behalf and on behalf of Power Test Realty Company Limited Partnership and Getty Properties Corp., the General Partner of Power Test Realty Company Limited Partnership

35. Legacy Vulcan, LLC / Vulcan Materials Company
36. Mallinckrodt LLC (f/k/a Mallinckrodt Inc.)
37. MI Holdings, Inc.
38. National-Standard, LLC
39. The Newark Group, Inc.
40. Newell Brands Inc. (f/k/a Newell Rubbermaid Inc.), on behalf of itself and its subsidiary Berol Corporation (as successor by merger to Faber-Castell Corporation)
41. Nokia of America Corporation (f/k/a Lucent Technologies Inc. / Alcatel-Lucent USA Inc.)
42. Novelis Corporation (Alcan Corp.)
43. Noveon Hilton Davis, Inc.
44. The Okonite Company, Inc.
45. Otis Elevator Company
46. Pabst Brewing Company, LLC
47. Pharmacia LLC
48. PPG Industries, Inc.
49. Public Service Electric and Gas Company
50. Purdue Pharma Technologies, Inc. (and Nappwood Land Corporation)
51. Quala Systems, Inc. and Quality Carriers, Inc.
52. Revere Smelting and Refining Corporation
53. Royce Associates, a Limited Partnership
54. Safety-Kleen Envirosystems Company, by McKesson Corporation, and McKesson Corp. for itself
55. Sequa Corporation
56. The Sherwin-Williams Company
57. Stanley Black & Decker, Inc.
58. STWB Inc.
59. Sun Chemical Corporation
60. TPL Management Operations, a series of Evergreen Resources Group, LLC on behalf of itself and Energy Transfer (R&M), LLC f/k/a Sunoco (R&M), LLC and Sunoco Partners Marketing & Terminals L.P.
61. Primary Products Ingredients Americas LLC (f/k/a Tate & Lyle Ingredients Americas LLC f/k/a A.E. Staley Manufacturing Company)
62. Textron, Inc.
63. United States Steel Corporation

Exhibit A

REPORT OF
DR. LINDA S. BIRNBAUM

Prepared for:
Preti Flaherty on behalf of the Small Parties Group

Prepared by:
Linda S. Birnbaum, PhD, DABT, ATS

March 21, 2023

1. Introduction

I, Linda S. Birnbaum, PhD, DABT, ATS, am a board-certified toxicologist with over forty years of experience serving as a federal scientist primarily studying environmental and public health, toxicology, and risk assessment. I am certified by both the American Board of Toxicology and the Academy of Toxicological Sciences. I received my MS and PhD in microbiology from the University of Illinois at Urbana-Champaign in 1969 and 1972, respectively. This report is based on my years of experience researching 2,3,7,8-TCDD and related compounds, my familiarity with human risk assessment, and the literature related to 2,3,7,8-TCDD's toxicity and effects on wildlife, experimental animals, cells in culture, and humans.[1]

For nineteen years, I directed the largest environmental health research division at the U.S. Environmental Protection Agency. Later, from 2009 to 2019, I served as the Director of the U.S. National Institute of Environmental Health Sciences of the National Institute of Health and the National Toxicology Program of the Department of Health and Human Services. I have also served as the Vice President of the International Union of Toxicology, the umbrella organization for toxicology societies in over fifty countries, and the President of the Society of Toxicology, the largest professional organization of toxicologists in the world. I was also the Chair of the Division of Toxicology of the American Society of Pharmacology and Experimental Therapeutics.

Currently, I am a Scientist Emeritus at the NIEHS and NTP and an adjunct professor at the Gillings School of Global Public Health, the Curriculum in Toxicology, and the Department of Environmental Sciences and Engineering at the University of North Carolina at Chapel Hill; at the Integrated Toxicology Program at Duke University; and at the Yale University School of Public Health. I am also a Scholar in Residence in the Nicholas School of the Environment of Duke University.

Over the past forty-plus years, I have authored more than one thousand peer-reviewed publications, book chapters, and reports. My research focuses on pharmacokinetic behavior of

[1] *See, e.g.*, Birnbaum, L.S., et al., *Toxic interaction of specific polychlorinated biphenyls and 2,3,7,8-tetrachlorodibenzo-p-dioxin: Increased incidence of cleft palate in mice*, Toxicol. & App. Pharma. 77(2):292-302 (1985); Birnbaum, L.S., *Distribution and excretion of 2,3,7,8-tetrachlorodibenzo-p-dioxin in congenic strains of mice which differ at the Ah locus*, Drug Meta. & Disp. 14(1):34-40 (1986); Birnbaum, L.S., et al., *Differential Toxicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) in C57BL/6J Mice Congenic at the Ah Locus*, Toxicol. Scis. 15(1):186-200 (1990); Birnbaum, L.S., *EPA's Reassessment of Dioxin Risk: Directed Health Research*, Chemosphere 27(1-3):469-475 (1993); Birnbaum, L.S., *The Mechanism of Dioxin Toxicity: Relationship to Risk Assessment*, Envtl. Health Persp. 102(9):157-167 (1994); Birnbaum, L.S., *Endocrine Effects of Prenatal Exposure To PCBs, Dioxins, and Other Xenobiotics*, Envtl. Health Persp. 102(8):676-679 (1994); Birnbaum, L.S. & DeVito, M.J., *Use of Toxic Equivalency Factors for Risk Assessment for Dioxins and Related Compounds*, Toxicology 105(2-03):391-401 (1995); Birnbaum, L.S., *Workshop on Perinatal Exposure to Dioxin-like Compounds v. Immunologic Effects*, Envtl. Health Persp. 103(supp. 2):157-160 (1995) Birnbaum, L.S., *Developmental Effects of Dioxins*, in REPRODUCTIVE & DEVELOPMENTAL TOXICOLOGY (1st ed. 1998); Birnbaum LS, *Dioxin and the AH receptor: Synergy of discovery*, Curr. Opinion in Toxicol. 2:120-123 (2017).

environmental chemicals, mechanisms of action of toxicants including endocrine disruption, and linking of real world exposures to health effects.

My work in toxicology has been recognized by the Mildred S. Christian Career Achievement Award from the Academy of Toxicological Sciences, the Society of Toxicology Distinguished Toxicology Scholar and SOT's Merit Award, multiple Scientific and Technological Achievement Awards from EPA, the NIH Director's Award, election to the National Academy of Medicine and as an AAAS Fellow, The North Carolina Science Award, and several honorary doctorates, among others. I am a recognized global expert on the health effects and risk assessment of dioxins. In the last three decades, I have been invited to participate in nearly all of the national and international assessments of dioxins that have taken place.

My full CV is provided in Appendix A.

2. Task

Based on my scientific knowledge, training, and experience, I have been asked to consider and give my conclusions on:

- The toxicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin ("2,3,7,8-TCDD" or "TCDD" or "dioxin") to humans.
- The scientific evidence and consensus regarding the health effects of 2,3,7,8-TCDD to humans.

3. Conclusions

a. 2,3,7,8-TCDD is the most toxic man-made substance known. It poses a significant threat to human health even at the lowest levels of exposure.

TCDD is the most toxic man-made compound. It is one compound in a family of structurally similar chemicals that have chlorine atoms at different positions on the benzene rings. There are 209 polychlorinated dioxins and furans: seventeen of these that have a common toxic mechanism of action and induce a common spectrum of biological responses in people, animals, and human and animal cells in culture. Because of the common toxic initiating mechanism of action, activation of the Ah receptor, dioxins and furans are analyzed and regulated as a group. TCDD is the most toxic of these related chemicals and is the chemical against which the toxicity of the other compounds is measured.[2]

Dioxins exist and are regulated as mixtures of dioxin-like compounds that have a common toxic mechanism of action, are structurally related, persistent, and cause similar biological responses in humans and animals. The U.S. Environmental Protection Agency ("EPA") regulates dioxins based on the WHO Toxic Equivalency Factor ("TEF") approach, which assigns each dioxin-like compounds a TEF value based on how toxic the compound is compared to TCDD. The TEF approach was initially proposed by dioxin researchers in the 1980s, and EPA recommended

[2] *See* U.S. EPA, *Recommended Toxicity Equivalence Factors for Human Health Risk Assessments of 2,3,7,8 TCDD and Dioxin-like Compounds*, at 13 (2010).

using the TEF Approach for evaluating human health risks from dioxins in 1987.[3] The first global consensus TEF values were published by the WHO in 1993.[4] EPA has continued to recommend using the WHO TEF values across human and ecological risk assessments.[5]

The TEF approach has been re-evaluated by the WHO's expert panel regularly, including in 1998, 2005, and 2022, and each time, the experts have reviewed new developments in the toxicology and public health literature and reaffirmed the basic approach. Each reevaluation has also evaluated the individual TEF values given to each dioxin-like compound and updated some of the relative TEF values for specific compounds as appropriate. Across each reevaluation, the status of TCDD as the benchmark toxic chemical has never changed, nor been undermined. The most recent reevaluation undertaken in 2022 has reaffirmed the toxicity of TCDD.[6]

The compounds evaluated using the TEF approach include dioxins, furans, and PCBs. Dioxins and certain PCB congeners cause similar biological effects but, structurally, they differ in the number and/or location of the chlorine atoms. Additionally, dioxins have two oxygen bridges connecting the benzene rings; furans have one oxygen bridge; and PCBs have no oxygen bridging the benzene rings. The oxygen bridges impart rigidity and planarity to the 3D structure.

Across the WHO reevaluations of TEF values, some PCBs have been added and later removed due to evolving understanding of PCBs' toxic modes of action.[7] The most dioxin-like and toxic PCB—PCB-126—has a TEF of 0.1, meaning it is ten times less toxic than TCDD. At times, researchers have used PCB-126 as an index compound to evaluate the relative toxicity of other PCBs, but even that calculation ultimately uses the comparison of PCB-126 to TCDD to establish the relative toxicity.[8] The most up-to-date reevaluation of dioxin-like compound TEF values did not increase the relative toxicity of PCBs compared to 2,3,7,8-TCDD.[9]

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[3] U.S. EPA, *Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs)* (1987).

[4] Ahlborg, U.G., et al. *Toxic Equivalency Factors for Dioxin-Like PCBs: Report on WHO-ECEH and IPCS Consultation*, Chemosphere (1994).

[5] U.S. EPA, *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife* (1993); U.S. EPA, *Supplementary guidance for conducting health risk assessment of chemical mixtures* (2000); U.S. EPA, *Exposure and human health reassessment for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds* (2003); U.S. EPA, *Analyses of Laboratory and Field Studies of Reproductive Toxicity in Birds Exposed to Dioxin-like Compounds for Use in Ecological Risk Assessment* (2003); U.S. EPA, *Framework for application of the toxicity equivalence methodology for polychlorinated dioxins, furans, and biphenyls in ecological risk assessment* (2008); U.S. EPA, *Recommended Toxicity Equivalence Factors for Human Health Risk Assessments of 2,3,7,8 TCDD and Dioxin-like Compounds* (2010).

[6] Personal communication.

[7] *See* Van den Berg, et al., *Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife*, Environ. Health Perspect. (1998).

[8] *See* Haws et al., *Development of a refined database of mammalian relative potency estimates for dioxin-like compounds*, Toxicol. Sci. (2006).

[9] Personal communication.

b. There is overwhelming scientific consensus on the fundamental toxicity of dioxins and 2,3,7,8-TCDD. TCDD is a human carcinogen and causes significant noncancer health effects including reproductive and developmental effects, immune suppression, hepatic, renal, bone, and skin effects, among others. Multigenerational effects have been observed in children.

There is scientific consensus that TCDD and dioxin-like compounds cause adverse effects in humans through activation of the aryl hydrocarbon receptor (AhR). The AhR is a highly conserved ubiquitous protein which binds with high affinity to dioxin in vertebrates. It is considered one of the key regulatory proteins that serves as a biological sensor for environmental stimuli and leads to the initiation of multiple biological processes. The triggering of AhR activity by TCDD causes and leads to a number of adverse health effects in humans, including cancer, cardiovascular disease, diabetes, porphyria, endometriosis, early menopause, reduced testosterone and thyroid hormones, altered immunologic response, skin, tooth, and nail abnormalities, altered growth factor signaling, and altered metabolism, among others.[10] Moreover, there is significant scientific consensus that there is no safe low dose of TCDD.[11]

i. Multi-Site Human Carcinogen

The longstanding, broad national and international scientific understanding is that TCDD is a multi-site human carcinogen. Evaluation of dioxin-like compounds and the health effects of TCDD by government agencies and other authorities has consistently identified TCDD as a human carcinogen. Below is a partial chronology of instances in which various agencies and authorities have characterized TCDD as or assessed or affirmed TCDD to be a human carcinogen:

- 1985 – EPA's initial TCDD guidance classified TCDD as a probable human carcinogen.[12]

- 1997 – International Agency for Research on Cancer ("IARC") (as a known human carcinogen).[13]

[10] White, S.S., & Birnbaum, L.S., *An overview of the effects of dioxins and dioxin-like compounds on vertebrates, as documented in human and ecological epidemiology, J. Envtl. Health & Sci. Part C: Envtl. Carcinogenesis & Ecotoxicology Revs.* (2009).

[11] The scientific consensus is that low dose linearity is possible when adding to a background rate. Crump, K.S., Crockett, P., *Improved confidence limits for low-dose carcinogenic risk assessment from animal data.* J Haz Matr. (1985); *see generally* Crump, K.S., *Use of threshold and mode of action in risk assessment,* Crit. Rev. Toxicol. (2011). A meta-analysis of three cohorts occupationally exposed to TCDD found a statistically significant trend in total cancer mortality with increasing dioxin exposure. Crump, K.S., et al., *Meta-analysis of Dioxin Cancer Dose Response for Three Occupational Cohorts.* Envtl. Health Perspect. (2003). A linear dose response provided a good fit to the combined data and predicted dioxin exposure resulting in a .01 increase in lifetime risk of cancer mortality of 45 pg/kg/day at a 95% confidence interval.

[12] U.S. EPA, *Health Assessment Document for Polychlorinated Dibenzo-p-Dioxin* (1985).

[13] World Health Organization, *IARC monographs on the evaluation of the carcinogenic risk to humans: Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans.* (1997).

- 2001 – The U.S. National Toxicology Program ("NTP") (as a known human carcinogen).[14]
- 2004 – EPA's draft reassessment (reaffirmed as a carcinogenic hazard).[15]
- 2004 – Stockholm Convention (186 participating countries).[16]
- 2006 – WHO's Dioxin Reevaluation.[17]
- 2010 – EPA's TEF Recommendation.[18]
- 2012 – EPA's Reanalysis of Dioxin Toxicity.[19]
- 2012 – IARC.[20]
- 2018 – European Food Safety Authority ("EFSA").[21]

Based on the study of cancer incidence among humans exposed to dioxins, EPA has, overall, concluded that there is a clear association between 2,3,7,8-TCDD and soft tissue sarcomas, lymphomas, and stomach carcinomas.[22]

IARC and the NTP have designated TCDD as a known human carcinogen based on limited epidemiological evidence, sufficient animal evidence, and mechanistic plausibility.[23, 24] The Institute of Medicine ("IOM") also determined that epidemiological evidence is sufficient to conclude that a positive association exists between exposure to herbicides contaminated with TCDD and increased risk for soft tissue sarcomas, B-cell lymphomas (Hodgkin lymphoma, non-Hodgkin lymphomas, chronic lymphocytic leukemia, hairy-cell leukemia), and monoclonal

[14] U.S. National Toxicology Program, *Report on Carcinogens* (2001).
[15] U.S. EPA, *Exposure and Human Health Reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin and Related Compounds* (2004).
[16] United Nations Environment Programme, *Stockholm Convention on Persistent Organic Pollutants* (2019).
[17] Van den Berg, M., et al., *Review: The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds*, Tox. Sci. (2006).
[18] U.S. EPA, *Recommended Toxicity Equivalence Factors (TEFs) for Human Health Risk Assessments of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Dioxin-Like Compounds.* (2010).
[19] U.S. EPA, *EPA's Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS Comments, Volume 1*. (2012).
[20] World Health Organization, *IARC monographs on the evaluation of the carcinogenic risk to humans: 2,3,7,8-Tetrachlorodibenzo-para-dioxin, 2,3,4,7,8-Pentachlorodibenzofuran, and 3,3',4,4',5-Pentachlorobiphenyl* (2012).
[21] European Food Safety Authority, *Risk for animal and human health related to the presence of dioxins and dioxin-like PCBs in feed and food*. (2018).
[22] U.S. EPA, *2,3,7,8-Tetrachlorodibenzo-p-Dioxin Hazard Summary*. (2000).
[23] World Health Organization, *IARC monographs on the evaluation of the carcinogenic risk to humans: Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans*. (1997).
[24] National Toxicology, P., *Report on Carcinogens, Fourteenth Edition* 2016.

gammopathy of undetermined significance.[25] In reaching these conclusions, IARC, NTP, and IOM reviewed the approximately thirty published studies examining the relationship between TCDD and increased risk of cancer. All of these studies shared a common thread: TCDD exposure causes increased risks of cancer.

Among those approximately thirty published studies are well-known, long-term studies focused on monitoring the health and mortality of groups of people who experienced occupational exposure to TCDD. For example, the National Institute for Occupational Safety & Health ("NIOSH") conducted a study of the mortality of 5,172 male workers at twelve plants in the United States that produced chemicals contaminated with TCDD. [26] This study included a subcohort of workers with a year or more of exposure to processes involving TCDD contamination and at least twenty years of latency. The NIOSH study concluded that the 5,172 male workers exposed to TCDD died from cancer at a 15% higher rate than the general population in the United States. With regard to the subcohort with a year or more of exposure to TCDD and at least twenty years of latency, the NIOSH study concluded those workers had a 46% increase in all cancers combined and a 42% increase in respiratory cancers.

In another study, the authors analyzed the health of veterans that were involved in the aerial spraying of Agent Orange and other TCDD-contaminated herbicides in Vietnam from 1962 to 1971, known as Operation Ranch Hand. Among the Operation Ranch Hand veterans, cancer risk increased significantly in association with increased exposure to TCDD. Those veterans with the highest exposure to TCDD experienced significantly higher rates of all-site cancers.[27]

In yet another study, the Center for Chemical Workers' Health and the Hamburg State Department of Work, Health, & Social Affairs analyzed a cohort of 1,189 male herbicide and insecticide workers with exposure to TCDD.[28] This study found that, in comparison to Germany's general population, the 1,189 workers with exposure to TCDD died from cancer at a 40% higher rate than the rest of the population. The cohort also experienced significantly increased rates of lung, respiratory, rectal, hematopoietic, and lymphatic cancers.

In an important long-term study, researchers have meticulously tracked the mortality and cancer incidence of a population exposed to TCDD by an accident in the Seveso area of Italy in 1976. The researchers divided the population into four geographic areas: Zone A, Zone B, Zone R, and a Reference Zone. As the accident produced a toxic cloud that moved south, Zone A was the most heavily contaminated area directly south of the plant, Zone B was further south and had medium contamination, and Zone R was the surrounding area with low contamination levels. The Reference Zone was a non-contaminated territory just to the south of the contamination zone. The populations of Zones A, B, and R were compared to the control group population of the Reference Zone. The populations in Zones A and B demonstrated increased risks of lymphatic and

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[25] National Academies of Science, Institute of Medicine, *Veterans and Agent Orange: Update 2018* (2018).
[26] Fingerhut MA, et al. 1991. *Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin.* New Engl. J. Med. (1991).
[27] Michalek, *Diabetes and Cancer in Veterans of Operation Ranch Hand*, at 336 (2008).
[28] Flesch-Janys, D., et al. *Estimation of the Cumulated Exposure to Polychorinated Dibenszo-p-dioxins/furans and Standardized Mortality Ratio Analysis of Cancer Mortality by Dose in an Occupationally Exposed Cohort.* Environ. Health Perspect. (1998).

hematopoietic tissue neoplasms, soft-tissue sarcoma, and non-Hodgkin Lymphoma.[29] The women in Zones A and B demonstrated a dose response-increased risk for breast cancer as well as a statistically significant increase in all cancers, in agreement with the male occupational studies.[30] The overall conclusion of the study of the Seveso population is that TCDD exposure represents a carcinogenic hazard.

In sum, the conclusion of every unconflicted author to study the morbidity and mortality of people exposed to TCDD, including IARC, NTP, IOM, and NIOSH, is that TCDD is a human carcinogen.

ii. Non-Cancer Health Effects

In addition to being a human carcinogen, TCDD also causes a plethora of non-cancer human health effects and diseases, including but not limited to developmental, reproductive, immune, skin and bone, liver and kidney, cardiovascular, and pulmonary issues. *See supra* Section 2.b.

For example, in one study, the Occupational Medical & Health Protection Department of BASF tracked the mortality of exposed employees of BASF after a 1953 explosion of a trichlorophenol unit in Germany that caused a major TCDD exposure event.[31] The study population included people who assisted in demolishing the trichlorophenol unit and other cleanup activities. It found that those employees with the highest TCDD concentrations in their blood lipids experienced an 18% increase in overall illness. This group also experienced higher rates of infectious and parasitic disease, nervous system and sense organ disorders, upper respiratory tract infections, and neoplasms.

Exposure to TCDD during human development is of great concern. It has serious impacts that often occur at the lowest exposure concentrations. There is significant scientific consensus that TCDD exposure causes changes to early human development *in utero* and early life including: altered thyroid and immune status, altered neurobehavior at the level of hearing, psychomotor function, gender-related behaviors, altered cognition, dentition, and development of reproductive organs, delays in breast development, hormonal signaling, and growth. Exposure to TCDD during development also causes altered sex ratios among the exposed offspring.[32]

iii. Multigenerational Effects

A growing body of scientific research is showing that dioxin exposure to adults can have multigenerational effects, with measurable differences in a child's brain development based on the exposures of the child's parents. Research following the mass-exposure event in Seveso, Italy, *see*

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[29] Pesatori, A.C., et al., *Cancer incidence in the population exposed to dioxin after the "Seveso accident": twenty years of follow-up.* Envtl. Health (2009).

[30] Warner, M., et al., Dioxin Exposure and Cancer Risk in the Seveso Women's Health Study, Envtl. Health Perspect., Vol. 119, Issue 12 (2011); *see also* Warner, M., et al., Serum dioxin concentrations and breast cancer risk in the Seveso Women's Health Study. Envtl. Health Perspect., Vol. 110, Issue 7 (2002).

[31] Zober, A., M.G. Ott, & P. Messerer, *Morbidity follow up study of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) after a 1953 chemical reactor incident.* Occup. Envtl. Med. (1994).

[32] White, S.S., & Birnbaum, L.S. (2009).

supra Section 2.1, has demonstrated paternal impacts on male sperm quality. Additionally, multigeneration effects are being shown in children and grandchildren of those living near U.S. military airbases in Vietnam where high environmental exposures to 2,3,7,8-TCDD occurred via Agent Orange.

For example, a recent study of multigenerational effects in children living around the military airbase in Bien Hoa, Vietnam has shown that lower neurodevelopmental scores are associated with elevated levels of TCDD in breast milk and umbilical cord blood.[33] Neurodevelopmental outcomes include significantly higher autism spectrum rating scale scores based on TCDD exposure.[34] General motor developmental and learning scores tested at the ages of 2, 3, and 5 indicate that such deficits are long-term.[35]

The results from the Bien Hoa Airbase studies, in particular, are relevant to the Diamond Alkali Superfund Site because exposures to people living near that airbase occur through ingestion of local foods, primarily fish and poultry.[36] This is similar to the exposure scenario for the Passaic River used by EPA, which shows that nearly all risk for those living along the Passaic River comes from ingesting locally caught fish and crabs.

With regard to the Diamond Alkali Superfund Site, it should be noted that, rather than accepting the scientific community's overwhelming consensus on the toxicity of and risk from TCDD, Occidental Chemical Company and its indemnitors spent decades producing scientifically conflicted studies regarding TCDD. The hundreds of studies they funded over the years had essentially two overarching objectives: minimizing the toxicity of and risk from TCDD; and maximizing the toxicity of and risk from other contaminants found in the Passaic River. Fortunately, by all accounts, these efforts failed; and despite these studies, the safety standards for TCDD have only increased over the years.

4. Conclusion

In summary, TCDD is the most toxic man-made substance. It is at least ten times more toxic than the most toxic PCB and many thousands of times more toxic than the average PCB. TCDD poses a significant threat to human health even at the lowest levels of exposure. Based on long-term studies of people exposed to TCDD, the scientific community and governmental authorities around the world concluded decades ago that TCDD is a carcinogen and causes a plethora of other non-cancer effects. Despite OxyChem's efforts to manufacture doubt where none

[33] Ngoc Nghi, et al., *Dioxins and Nonortho PCBs in Breast Milk of Vietnamese Mothers Living in the Largest Hot Spot of Dioxin Contamination*, Envtl. Sci. Tech. 49(9):5732-5742 (2015); Boda et al., *Prenatal dioxin exposure estimated from dioxins in breast milk and sex hormone levels in umbilical cord blood in Vietnamese newborn infants*, Sci. of the Total Envt. 628:484-489 (2018).

[34] Ngoc Pham, et al., *Effect of Perinatal Dioxin Exposure Originating from Agent Orange on Gaze Behavior in 3-Year-Old Children Living in the Most Dioxin-Contaminated Areas in Vietnam*, Toxics 10(4) (2022).

[35] Ban Trang et al., *Adverse effects of dioxins on cognitive ability and motor performance of 5-year-old children residing in a hotspot of dioxin contamination originating from Agent Orange in Vietnam: A prospective cohort study*, Sci. of the Total Envt. 833 (2022).

[36] Hung Minh, et al., *Bioaccumulation of PCDD/Fs in foodstuffs near Bien Hoa and Da Nang airbases: assessment on sources and distribution*, Envtl., Sci. & Poll. Res. 26:28852-28859 (2019).

exists, recent health studies and evaluations by governmental authorities support and reaffirm these longstanding conclusions about the toxicity of and risk from TCDD.

Dated: 3/21/23

DR. LINDA S. BIRNBAUM, PHD, DABT, ATS

Linda S. Birnbaum

Appendix A

Linda S. Birnbaum, Ph.D., D.A.B.T., A.TS.

CURRICULUM VITAE

Last Updated March 2023

Date and Place of Birth: December 21, 1946; Passaic, New Jersey

Citizenship: United States

Marital Status: Married 1967, three children

Education

June 1967	B.A. (Biology) University of Rochester, Rochester, NY
June 1969	M.S. (Microbiology) University of Illinois, Urbana, IL
February 1972	Ph.D. (Microbiology, Biochemistry minor) University of Illinois, Urbana, IL (Thesis: Localization, Enrichment, and in vitro Transcription of Ribosomal RNA genes in Escherichia coli)
1982	Diplomate, American Board of Toxicology, recertified 1987, 1992, 1997, 2002, 2007, 2012, 2018,2023

Brief Chronology of Employment

1972	Visiting Assistant Professor of Microbiology at University of Illinois, Urbana, IL
1973 - 1974	Postdoctoral work at University of Massachusetts, Amherst, MA (Biochemistry)
1974 - 1975	Assistant Professor of Science at Kirkland (Hamilton) College, Clinton, NY
1975 - 1976	Research Associate, Masonic Medical Research Laboratory, Utica, NY
1976 - 1978	Research Fellow, Masonic Medical Research Laboratory, Utica, NY
1978 - 1979	Research Scientist, Masonic Medical Research Laboratory, Utica, NY
1979 - 1980	Senior Staff Fellow, National Toxicology Program, National Cancer Institute, Research Triangle Park, NC
1980 - 1987	Research Microbiologist, National Toxicology Program, NIEHS, Research Triangle Park, NC
1987 - 1989	Supervisory Research Microbiologist, National Toxicology Program, NIEHS, Research Triangle Park, NC
1989 - 1989	Head, Chemical Disposition Group, NIEHS, Research Triangle Park, NC
1989 - 1998	Director, Experimental Toxicology Division, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC
1998 - 1998	Acting Associate Director for Health, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC
1999 - 2008	Director, Experimental Toxicology Division, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC
2001 - 2002	Acting Director, Human Studies Division, National Health and Environmental Effects Research Laboratory, U.S. EPA, Chapel Hill, NC

Appendix A

2002 - 2009	Director, Experimental Toxicology Division, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC
2006- Present	Fellow, June 2006-June 2011; Board member, July 2007-June 2010, Academy of Toxicological Sciences
2008 - 2009	Senior Toxicologist, National Center for Environmental Assessment, U.S. EPA, Research Triangle Park, NC
2009 –2019	Director, National Institute of Environmental Health Sciences and National Toxicology Program, Research Triangle Park, NC
2009 - 2019	Senior Investigator, National Cancer Institute
2019 - Present	Scientist Emeritus, Division of Translational Toxicology, NIEHS
2020 – Present	Scholar in Residence, Nicholas School of the Environment, Duke University

Societies

North Carolina Chapter, Society of Toxicology

Society of Toxicology (President 2004-2005)

American Society for Pharmacology and Experimental Therapeutics

ASPET Division of Toxicology (Chair, 1997-1998)

American Board of Toxicology

American Association for the Advancement of Science

Phi Beta Kappa

Phi Kappa Phi

Sigma X

Academy of Toxicological Sciences

International Union of Toxicology (Vice President 2007-2009)

Society for Risk Analysis

National Academy of Medicine

Interagency Autism Coordinating Committee

Toxicology Forum

Appendix A

Awards

1967-1972	NIH Predoctoral Traineeship
1971	Sigma Xi Award, University of Illinois
1973-1974	Damon Runyon Foundation Postdoctoral Fellowship
1974-1975	Mellon Foundation Research Grant
1976-1978	National Research Service Award
1978-1979	Career Employment and Training Award
1979	Young Investigator Grant - N.I.A.
1991	Scientific and Technological Achievement Award, Level III, U.S EPA
1992	Scientific and Technological Achievement Award, (2 awards) Level III, U.S. EPA
1993	Scientific and Technological Achievement Award, Level III, U.S. EPA
1994	Scientific and Technological Achievement Award, Level III, U.S. EPA (2 awards)
1996	National Wildlife Federation, Special Edition National Conservation Achievement Award
1996	First Ahlborg Memorial Award, Karolinska Institute, Sweden
1998	Scientific and Technological Achievement Award, Level III, U.S. EPA
1999	Best Risk Assessment Paper, SOT, New Orleans, LA
1999	Scientific and Technological Achievement Award, Level II, U.S. EPA
2000	Scientific and Technological Achievement Award, Honorable Mention
2000	Scientific and Technological Achievement Award, Honorable Mention
2001	Diversity Leadership Award, U.S. EPA
2002	Recognized as one of the top 100 cited authors in ISI Pharmacology
2002	U.S. EPA Gold Award for Scientific Achievement in the Health Sciences
2003	Society of Toxicology Risk Assessment Specialty Section (RASS) Blue Ribbon, best abstract
2003	Scientific and Technological Achievement Award, Level II, U.S. EPA
2004	U.S. EPA Bronze Medal (Region 5) to Emerging Pollutants Workshop Planning Group
2004	Society of Toxicology Risk Assessment Specialty Section (RASS) Blue Ribbon, best abstract
2004	Scientific and Technological Achievement Award, Level III, U.S. EPA
2005	Scientific and Technological Achievement Award, Level III, U.S. EPA
2006	Academy of Toxicological Sciences, Fellow
2006	Mid-Atlantic Chapter of the Society of Toxicology (MASOT) Ambassador Award
2006	Environmental Science & Technology Excellence in Review
2006	Society of Toxicology Public Communications Award
2007 - Present	Society of Toxicology Endowment Board
2007 - 2010	President-Elect/President for International Union of Toxicology (IUTOX)
2007 - Present	Official Advisor to the Endometriosis Association
2007 - Present	Editorial Board, Environmental Health Perspectives
2007	Scientific and Technological Achievement Award, Level I, U.S. EPA
2007	Scientific and Technological Achievement Award, Level III, U.S. EPA
2008	Society of Toxicology (SOT) 2008 Women in Toxicology (WIT) Elsevier Mentoring Award
2010	Elected to the Collegium Ramazzini

Appendix A

2010	Honorary Doctorate from the University of Rochester
2010	College of Liberal Arts & Science Alumni Achievement Award, University of Illinois
2010	Elected to the Institute of Medicine of the National Academies of Science
2011	National Institutes of Health Director's Award
2011	Scientific and Technological Achievement Award, Level III, U.S. EPA
2012	Breast Cancer Fund Heroes Award
2012	National Research Center for Women's 2012 Health Policy Hero Award
2012	Scientific and Technological Achievement Award, Level III, U.S. EPA
2012	NIH Children's Environmental Health Network Child Health Science Advocate Honoree
2013	American Public Health Service Homer N. Calver Lecturer Award
2014	Mailman School of Public Health, Columbia University, Granville H. Sewell Distinguished Lecturer
2014	Honorary Doctorate from Ben-Gurion University, Israel
2014	Surgeon General's Medallion 2014
2014	EPA ORD Impact Award: Children's Environmental Health and Disease Prevention Research Centers
2014	National Institutes of Health Director's Award
2015	Honorary Professor, University of Queensland, Australia
2016	North Carolina Award for Science
2016	NIEHS Champion of Environmental Health Research
2017	Society of Toxicology Distinguished Toxicology Scholar Award
2017	Honorary Doctorate from the Amity University, India
2018	Arnold J. Lehman Award, Society of Toxicology
2018	Mildred S. Christian Career Achievement Award, Academy of Toxicological Sciences
2020	Frank Hatch Environmental Health Leadership Award, Defend Our Health
2021	Honorary Doctor of Science from the University of Rhode Island
2021	2021 Ramazzini Award, Collegium Ramazzini
2022	Society of Toxicology Merit Award
2022	Annual PFAS Meeting Lifetime Achievement Award
2022	Elected AAAS Fellow

Appendix A

Other Activities

1976	Adjunct Professor, Genetics, State University College of Technology, Utica, NY
1977 - 1979	Chairperson, Guest Speaker Program at Masonic Medical Research Laboratory
1978 - 1979	Consultant, Syracuse Research Corporation (detection of carcinogens as mutagens)
1979 - 1983	Member, Executive Board, American Aging Association
1980 - 1981	Vice President, American Aging Association
1980 - Present	Adjunct Professor, Department of Environmental Science, School of Public Health, University of North Carolina
1983 – Present	Curriculum in Toxicology, University of North Carolina
1985 – 1988	Editorial Board, AGE
1988 – 1994	Editorial Board, Environmental Health Perspectives
1989 – 1993	Editorial Board, Toxicology and Applied Pharmacology
1989 – 1993	Executive Committee, Curriculum in Toxicology, University of North Carolina
1992 – 1996	Member of the Chemical Manufacturers Association Butadiene Panel
1993 – 1998	Editorial Board, Environmental Health Perspectives
1993 – 1995	External Advisory Co Committee for NIEHS Planning Grant for an EHS Center
1993 – 2009	Editorial Board, Human and Experimental Toxicology
1994 – 1997	Elected to serve a three-year term on the Executive Committee of the Division of Toxicology, ASPET
1994 – Present	Reviewer for Medical Research Council of Canada Grants
1995 – 1998	CIIT Scientific Advisory Panel
1995 – Present	Adjunct Professor of Toxicology, Integrated Toxicology Program, Duke University
1999 – 2000	Chair, Division of Toxicology, ASPET
1999 – 2009	Editorial Board, Chemosphere
2000 – 2007	Executive Committee, Research Triangle Park Drug Metabolism Discussion Group
2000 – 2004	U.S. Delegate to AMAP/Health Effects Group
2002 – 2006	Board of Directors of SOT as Vice President Elect, Vice President, President, and Past President
2013 – Present	Editorial Board, Environment International
2016 – Present	Editorial Board, Current Opinions in Toxicology
2019 – Present	International Advisory Board Member, INSERM, French National Institute of Health and Medical Research
2021 – Present	International Organizing Committee, Dioxin2022
2021 – Present	International Organizing Committee, BFR2022
2021 – 2026	Professor Adjunct of Epidemiology, Department of Environmental Health Sciences, Yale School of Public Health

Invited Speaker

Liver and Aging, II (Japan, 1982)

SOT (Atlanta, 1984)

Organizer, Annual symposium, American Aging Association (1984)

Appendix A

Butadiene ISSRP Workshop (1985)

Toxicity and Aging Workshop (EPA/NIA, 1985)

Gerontology Society (New Orleans, 1985)

Strain Selection for Carcinogenesis (NIEHS, 1985)

Gerontology Society (Chicago, 1986)

Dioxin '87 (Las Vegas, 1987)

Pharmacokinetics Modeling and Risk Assessment (Asheville, NC, 1988)

ASPET (Montreal, 1988); WHO Workshop on Chemical Toxicity and Aging (Leningrad, 1988)

SOT (Atlanta, 1988)

FASEB (New Orleans, 1989)

North Carolina Academy of Sciences (Raleigh, 1989)

Human Health Effects of Pollution in the Great Lakes (Ontario, 1989)

NCASI Dioxin Research Needs Expert Panel (Rockville, MD,1989)

15th Symposium Environmental Pollutants and Toxicology (Japan, 1989)

PCB Workshop, Health Protection Branch, Health and Welfare (Ottawa, Canada, 1990)

International Cancer Congress (Hamburg, West Germany, 1990)

TEFs for PCBs (Washington, DC, 1990)

Women Administrators in North Carolina Higher Education Spring Forum meeting on "Women in Science" (Durham, NC, 1992)

State of North Carolina, Department of Environment, Health and Natural Resources, Division of Environmental Management Commission's Water Quality Committee (Raleigh, NC, 1992)

SEGH International Conference on Lead and Other Trace Substances (1992)

Rifkin and Associates "Mechanisms of Dioxin Toxicity: Implications for Risk Assessment (Washington, D.C. 1993)

California Department of Health Services, Hazardous Materials Laboratory (Berkeley, California 1993)

Gordon Research Conference, "Dioxin Toxicity and Risk Assessment" (Meriden, New Hampshire, 1993)

American College of Toxicology (1993)

Health Protection Branch, Health and Welfare, Canada (1993)

Butadiene Peer Review Panel (1993)

HERL First Annual Symposium (1993)

Waste Technologies Incorporated Peer Review Workshop (1993)

North Carolina State Graduate Student Professional Development Workshop (1993)

HERL Symposium (1993)

NIEHS Sponsored Estrogens in the Environment III: Global Health Implications (1994)

National Symposium on Health Research and Needs to Ensure Environmental Justice (1994)

AEERL/ASME Seminar on PIC Formation and Control (1994)

Children's Environmental Health Network (1994)

Appendix A

EPA Colloquium on Environmental Hormones (1994)

NIEHS Second Annual Environmental Careers Symposium (1994)

Chemical Manufacturers Association, Chlorine Chemistry Council (1994)

University of Pittsburgh (1994)

Regional Risk Assessment Meeting (1994)

Great Lakes Human Health Effects Research Symposium (1994)

European Environmental Research Organization (1994)

Pennsylvania State 13th Summer Symposium in Molecular Biology (1994)

Rifkin & Associates (1994); 6th North American ISSX Meeting (1994)

HERL Second Annual Symposium (1994)

American Zoological Society (1995)

Mechanistic Studies - National Toxicology Program (1995)

Chlorination and Drinking Water; Dioxin Roundtable - SOT (1995)

International Joint Commission - Great Lake's Science Advisory Board (1995)

WHO (1995)

Freie University of Berlin (1995)

International Institute of Synthetic Rubber Producers, Inc. (1995)

International Congress of Toxicology (1995)

Working Group on the Assessment of Health Risk for Infants from Exposure to PCDDs, PCDFs, and PCBs (1995)

International Neurotoxicology Conference (1995)

Endometriosis Association (1995)

PCB Assessment Panel (1996)

Dioxin Risk Characterization Working Team (1996)

International Symposium of the Society of Toxicologic Pathologists (1996)

Ulf G. Ahlborg Memorial Lecture at Karolinska Institutes Nobel Forum (1996)

Russian-American Project with scientists, policymakers and citizen Stakeholder Uniting to Reduce dioxin levels in the Environment and Human Beings, Russia (1996)

Dioxins in the Middle East Workshop, Israel (1996)

69th Meeting of the IARC Monographs Programme on the Evaluation of Carcinogenic Risks to Humans, France (1997)

WHO Workshop on Revision of I-TEFs, Sweden (1997)

Pellston/SETAC Workshop on Chemical Effects on Reproduction, Montana (1997)

ISSX Meeting, SC (1997)

WHO Workshop on TDI for Dioxins (1998)

CIIT Workshop on PBPK Modeling of 1,3-Butadiene (1998)

Dioxin1998, Sweden (1998); Autoimmune Disease Workshop (1998); FQPA Meeting (1999)

Workshop on Steroid Hormones & Brain Function, Breckenridge, CO (1999)

Appendix A

Drinking Water Research Needs Expert Workshop, Leesburg, VA (1999)

Human Exposure Assessment Workshop, Rockville, MD (1999)

College of Pharmacy, Washington State University, Pullman, WA (1999)

Symposium on Man-Made Chemicals/Hormones in the Environmental on Human Health, Mt. Holyoke College, South Hadley, MA (1999)

IPCS Planning Group Meeting on Integrated Risk Assessment, Philadelphia, PA (1999)

13th Working Group Meeting of the Arctic Monitoring and Assessment Program, Toronto, Canada (1999)

Workshop on Criteria for Phasing Out Persistent and Bioaccumulating Organic Chemicals, Stockholm, Sweden (1999)

Workshop on Diversity 2000: Managing the Third Wave (1999)

Endocrine Disruptors and Children's Health, Mt. Sinai School of Medicine, New York (2000)

Living Safely with Chemicals in the New Millennium (2000)

AMAP Workshop, Rovaniemi, Finland (2000)

PAS Proteins/ASPET, Boston, MA (2000)

CMA Workshop Biomarkers, RTP, NC (2000)

ASPET Program Committee (2000)

WHO Integrated Risk Assessment Workshop, Ispra, Italy (2001)

Brominated Flame Retardants (BFR) Stockholm, Sweden (2001)

WHO Expert Meeting on Rapid Assays, Brussels, Belgium (2001)

Conference on Endocrine Disrupters and Human Health, Universidade Independente, Lisbon, Portugal (2001)

Vietnam-United States Scientific Conference on Human Health and Environmental Effects of Agent Orange/Dioxin, Hanoi, Vietnam (2002)

Federal-State Toxicology and Risk Analysis Committee, Washington, DC (2002)

European Commission Non-dioxin-like PCBs Workshop, Brussels, Belgium (2002)

Brominated Flame Retardants Roundtables, San Francisco, CA (2002)

Federal Women's Program, Women's History Month Event, Research Triangle Park, NC (2003)

Society of Toxicology Annual Meeting, Salt Lake City, Utah (2003)

Federal Women's Program, Women's History Month Event, Research Triangle Park, NC (2003) Brominated Flame Retardants, Conference & Workshop, San Francisco, CA (2003)

SAIC Conference on Dioxin Threat Assessment, McLean, VA (2003)

Star Progress Review Workshop, Washington, DC (2003)

Senior Executive Service Meeting, Washington, DC (2003)

Emerging Pollutants Workshop, Boston, MA (2003)

Dioxin Workshop, Boston, MA (2003)

Cocktail Effect in Risk Assessment (CeiRA), Stresa, Italy (2003)

PBDE Workgroup, Washington, DC (2004)

19th Annual Regional Risk Assessors Meeting, Boston, MA (2004)

Appendix A

U.S. EPA Region I Risk Assessors, Boston, MA (2004)

PCB Workshop, University of Illinois, Urbana-Champaign, Illinois (2004)

EPSA Science Colloquium, Brussels, Belgium (2004)

ICTX Satellite & ICTX Meeting, Porvoo, Finland (2004)

Dioxin2004, Berlin, Germany (2004)

HEI Biomonitoring, Research Triangle Park, NC (2004)

Bear Mountain Superfund, Bear Mountain, NY (2004)

Region IV ERS, Atlanta, GA (2004)

NRP 50 Bern, Switzerland (2004)

Environmental Influences on the Induction and Incidence of Asthma, Research Triangle Park, NC (2004)

CHPAC Washington, DC (2004)

APHA, Washington, DC (2004)

University of Michigan Dioxin Exposure Study (UMDES) SAB, Ann Arbor, MI (2004-2009)

CASCADE, Orvieto, Italy (2005)

Society of Toxicology Annual Meeting, New Orleans, Louisiana (2005)

Credo Workshop on Endocrine Disrupters, Prague, Czech Republic (2005)

TEFs Re-Evaluation, World Health Organization, Geneva, Switzerland, (2005)

1st International Workshop on Modifiers of Chemical Toxicity, Poros/Athens, Greece (2005)

Children's Environmental Health Research, Research Triangle Park, NC (2005)

Dioxin2005, Toronto, Canada, (2005)

42nd Congress of the Federation of European Toxicologists & European Societies of Toxicologists (EUROTOX2005), Krakow, Poland (2005)

National Forum on Contaminants in Fish, Baltimore, MD (2005)

Nicholas Institute for Environmental Policy Solutions, Inaugural, Duke University, Durham, NC (2005)

U.S. EPA Region II Science Day, New York, New York (2005)

40th Annual Meeting, American Academy of Environmental Medicine, Tucson, AZ (2005)

North Carolina Society of Toxicology Spring Meeting, 25th Anniversary Celebration, Research Triangle Park, NC (2006)

CASCADE 2nd Annual Meeting, St. Malo, France (2006)

Mid-Atlantic Society of Toxicology (MASOT) Meeting, Scotch Plains, New Jersey (2006)

Gordon Research Conferences, Environmental Endocrine Disruptors, Il Ciocco, Barga, Italy (2006)

28th International Congress on Occupational Health, Milan, Italy (2006)

International Brominated Flame Retardants (BFR) Meeting, Toronto, Canada (2006)

Bisphenol A: An Examination of the Relevance of Ecological, In Vitro and Laboratory Animal Studies for Assessing Risks to Human Health – An Expert Panel, Chapel Hill, NC, (2006)

Environmental Challenges Meeting, San Francisco, CA (2007)

UCSF-CHE Summit on Environmental Challenges to Reproductive Health and Fertility, San Francisco, CA (2007)

Appendix A

State-of-the-Science Workshop: Issues and Approaches in Low Dose-Response Extrapolation for Environmental Health Risk Assessment, Baltimore, MD (2007)

NERC Knowledge Transfer Network, 2nd Conference on Persistent Organic Pollutants: Legacy and Current-Use Pops, The University of Birmingham, Birmingham, UK (2007)

LTIG Meeting, Chicago, IL (2007)

Moving Upstream Workshop, Berkeley, CA (2007)

International Congress of Toxicology, Montreal, Quebec, Canada (2007)

Dioxin2007 Satellite Symposium, Shibuya-ku, Tokyo, Japan (2007)

Dioxin2007 International Symposium, Tokyo, Japan (2007)

International Congress of Toxicology XI, Montreal, Ontario, Canada (2007)

Future Research on Endocrine Disruption, Durham, NC (2007)

NHEERL/OSWER Libby Action Plan Toxicology Studies, OU4 Technical Subgroup Meeting, Libby, MT (2007)

SETAC North America 28th Annual Meeting, Milwaukee, WI (2007)

Society of Risk Analysis 2007 Annual Meeting, San Antonio, TX (2007)

Meeting of the HESI Subcommittee on Risk Assessment for Sensitive Populations, Washington, DC (2007)

47th Annual Society of Toxicology & ToxExpo, Seattle, Washington (2008)

6th Expert Consultation Panel Meeting for Provisional Advisory Levels, Research Triangle Park, NC (2008)

5th PCB Workshop, Iowa City, IA (2008)

10th GRC on EED, PFAA Days, Research Triangle Park, NC (2008)

Woman Taking the Lead to Save our Planet, NIH, Washington, D.C. (2009)

Annual Society of Toxicology Conference, Baltimore, MD (2009)

Columbia Center for Children's Environmental Health Conference, New York, NY (2009)

Brominated Flame Retardants (BFR), Annual Meeting, Ottawa, Canada (2009)

Institute of Medicine Environmental Roundtable Meeting (2009)

Green Chemistry Conference (2009)

National Conversation on Public Health and Chemical Exposures Kick-Off Meeting (2009)

Toxicology Forum (2009)

Developmental Basis for Disease Workshop (2009)

29th International Symposium on Halogenated Persistent Organic Pollutants at Dioxin (2009)

Keynote at the Rachel Carson Legacy Conference, Pittsburgh, PA (2009)

Plenary at ISES Conference, Minneapolis, MN (2009)

Annual NIEHS-NCI BCERC Conference, Oakland, CA (2009)

Thyroid Biomarkers Workshop, San Francisco, CA (2009)

Joint Workshop on Environmental Pollution and Cancer in China and the U.S., Guangzhou, China (2010)

University Research Corridor Conference (2010)

Annual Society of Toxicology Conference, Salt Lake City, UT (2010)

Appendix A

UVA Plastic Project Workshop (2010)

Children's Environmental Health Task Force Workshop (2010)

Teratology Society Annual Meeting (2010)

Genetics and Environmental Mutagenesis Society Meeting, RTP, NC (May 2011)

Health Affairs Environmental Challenges for Health, Washington, DC (May 2011)

Congressman Price Science Panel, RTP, NC (June 2011)

National Medical Association Annual Meeting, Washington, DC (July 2011)

Gordon Research Conference on Cellular & Molecular Mechanisms of Toxicity, Andover, MA (August 2011)

Brussels Fire Retardant Dilemma Symposium, Brussels, Belgium (August 2011)

GlaxoSmithKline Women in Science Annual Meeting, RTP, NC (October 2011)

Los Angeles Community Forum, CA (October 2011)

Duke Integrated Toxicology Environmental Health Program, Durham, NC (November 2011)

Medical University of South Carolina, Charleston, SC (January 2012)

Parkinson's Action Network Panel, Washington, DC (February 2012)

Texas Women's University Annual Celebration of Science, Denton, TX (March 2012)

UNC Women in Science Series, Chapel Hill, NC (March 2012)

Environmental Health Sciences Seminar, UC-Davis, CA (March 2012)

South Atlantic National Research Conference, Raleigh, NC (March 2012)

Society of Toxicology Annual Meeting: Session on Career and Meet the Directors, San Francisco, CA (March 2012)

PPTOX III, Session XI: Future Agenda & Conference Conclusions, Paris, France (May 2012)

EU Conference on Endocrine Disrupters, Seminar & Panelist, Brussels, Belgium (June 2012)

University of Rochester, Toxicology Retreat, Rochester, NY (May 2012)

ENDO 2012, Presidential Symposium, Houston, TX (June 2012)

32nd International Symposium on Halogenated Persistent Organic Pollutants (DIOXIN), Endocrine Disruptor Chemicals (Chair of session), Cairns, Australia (August 2012)

University of Michigan, Conference on the Developmental Origins of Metabolic Syndrome, MI (October 2012)

Pesticides & The Chesapeake Bay Watershed Project, Reisterstown, MD (October 2012)

Workshop: FutureTox: Building the Road for 21st Century Toxicology and Risk Assessment Practices, Arlington, VA (October 2012)

Association of Schools of Public Health Environmental and Occupational Health Council, San Francisco, CA (October 2012)

APHA, Gulf Oil Spill Session, San Francisco, CA (October 2012)

Breast Cancer and the Environment Research Program (BCERP) Annual Meeting, Keynote Address, San Francisco, CA (November 2012)

Climate Change, Workplace and the Lung Workshop, Keynote Address, Maulana Azad Medical College, New Delhi, India (December 2012)

Appendix A

EHS Research Retreat Global Environmental Health, Johns Hopkins, Baltimore, MD (January 2013)

Toxicology Forum Winter Meeting, Session on Low Dose Effects, Washington, DC (January 2013)

Collaborative Summit on Breast Cancer Research, Panelist, Washington, DC (February 2013)

Environmental Health 2013, Boston, MA (March 2013)

Society of Toxicology Annual Meeting: Session on Career and Meet the Directors, San Antonio, TX (March 2013)

13th International Congress on Combustion By-Products and Their Health Effects, New Orleans, LA (May 2013)

XIII International Congress of Toxicology, Genes and the Environment, Seoul, South Korea (July 2013)

National Environmental Monitoring Conference, Session on Low Dose Exposure of Environmental Chemicals, San Antonio, TX (August 2013)

45th Annual Symposium of the SOT of Canada, Plenary Speaker, Ottawa, Canada (December 2013)

Brunel University, Middlesex, United Kingdom: Keynote Speaker at a Roundtable on the Role of Chemicals in the Fetal Environment & Presenter of a Seminar at the Brunel University (February 2014)

Society of Toxicology Annual Meeting: Session on Career and Meet the Directors, Phoenix, AZ (March 2014)

Environment and Health Fund & Israeli Ministry of Health Meeting, Speaker, Hebrew University, Jerusalem, Israel (May 2014)

Tribal Environmental Health Summit: Building Collaborative Community Networks, Salish Kootenai College, Pablo, MT (June 2014)

Meeting on Women's Health sponsored by Representative Nita Lowey, Speaker, Mercy College, Dobbs Ferry, NY (July 2014)

Green Science Policy Meeting, Chair of Session on TBBPA and Presenting at Session on Integrating Toxicology & Epidemiology, Madrid, Spain (September 2014)

8th International PCB Workshop: PCBs in Schools, Presenter in Session on Anniston Community Health Survey, Woods Hole, MA (October 2014)

PPTOX IV, Opening Session, Boston, MA (October 2014)

Annual Biomedical Research Conference for Minority Students, Speaker at Professional Development Session, San Antonio, TX (November 2014)

APHA Annual Meeting, Speaker at Session on Gulf of Mexico Research Initiative: First 3 Years, New Orleans, LA (November 2014)

U.S. Environmental Protection Agency Cookstove Conference, Speaker on NIEHS Research on Cookstoves, RTP, NC (February 2015)

Tox21 Workshop and Bioassay Roundtable, Society of Toxicology, San Diego, CA (March 2015)

Targeting Environment and Neuro-Developmental Risks (TENDR), Warrenton, VA (June 2015)

Symposium on Endocrine Disrupting Chemicals, Tokyo, Japan (July 2015)

IX Congress of Toxicology in Developing Countries, XIX Congresso Brasileiro de Toxicologia, Natal, Brazil (November 2015)

International Society for Children's Environmental Health, Cuernavaca, Mexico (January 2016)

Appendix A

Society of Toxicology, New Orleans, LA (March 2016)

Invited Keynote Speaker, University of Wisconsin Madison Symposium: Toxicology and Urology (April 2016)

Speaker, Trans-NIH Transgenerational Inheritance Workshop (April 2016)

Opening Speaker at the NTP Workshop "Addressing Challenges in the Assessment of Botanical Dietary Supplement Safety," Bethesda, MD (April 2016)

Opening Remarks at TaRGET II Consortium Grantee Meeting, NIEHS, NC (May 2016)

Invited Speaker at the 2016 Tribal Environmental Health Summit, Flagstaff, AZ (June 2016)

Speaker, Appalachia/Kentucky Community Forum, Lexington & Hazard, Kentucky (July 2016)

Presented 5 talks at Dioxin 2016, Florence, Italy, and 2 talks at 28th Conference of the International Society for Environmental Epidemiology, Rome, Italy (August 2016)

Invited Speaker, Annual Regulatory Summit for American Home Furnishings Alliance, Hickory, NC (September 2016)

Speaker, Scientific Advisory Committee on Alternative Toxicological Methods (SACATM), NIEHS, NC (September 2016)

US Chapter of International Society for Developmental Origins of Health and Disease (DOHaD), Detroit, MI (October 2016)

The Korean Academy of Science and Technology (KAST), Seoul, Korea (November 2016)

Research and Policy Needs for Environmental Health Workshop, Israel Institute for Advanced Studies, Givat Ram, Israel (January 2017)

Society of Toxicology 56th Annual Meeting and ToxExpo Global Collaboration, Boston, MA (March 2017)

Society of Toxicology 56th Annual Meeting and ToxExpo Distinguished Toxicology Scholar Award Lecture, Boston, MA (March 2017)

US Senate Appropriations Subcommittee on Interior, Environment and Related Agencies Briefing on NIEHS Superfund Activities, Washington, DC (June 2017)

37th International Symposium on Halogenated Persistent Organic Pollutants - Dioxin 2017, Vancouver, British Columbia (August 2017)

Collegium Ramazzini Days, Carpi, Italy (October 2017)

Keynote Speaker at the 2017 American College of Toxicology Annual Meeting, Palm Springs, CA (November 2017)

12th Annual Breast Cancer and the Environment Research Program Meeting, Monrovia, CA (November 2017)

Keynote Speaker at the International Conference on Impact of Environment on Women's Health, Lucknow, India (November 2017)

Keynote Speaker at the Environmental and Health Fund Annual Conference, Jerusalem, Israel (December 2017)

Keynote Speaker at the American Academy of Allergy, Asthma and Immunology Meeting, Orlando, FL (March 2018)

Society of Toxicology Annual Meeting and Tox Expo, San Antonio, TX (March 2018)

Keynote Speaker at Baylor College of Medicine, Houston, TX (April 2018)

Toxicology and Risk Assessment Conference, Cincinnati, OH (April 2018)

Keynote Speaker at International Conference on Medicine One Science (ICOMOS), Minneapolis, MN (April 2018)

Closing Plenary at Tribal Environmental Health Summit at Oregon State University, Corvallis, Oregon (June 2018)

Appendix A

EHS Core Center and Community Forum, Davis, CA (July 2018)

American Association for Clinical Chemistry Annual Meeting and Clinical Lab Expo, Chicago, IL (August 2018)

ISEE/ISES Conference, Ottawa, Canada (August 2018)

Federal Hearing: The Federal Role in the Toxic PFAS Chemical Crisis, Washington, DC (September 2018)

North Carolina Central University Class Lecture, Durham, NC (October 2018)

ExxonMobil Biomedical Sciences, Inc., Annandale, NJ (October 2018)

National Academy of Medicine's 48th Annual Meeting, Washington, DC (October 2018)

University of Texas El Paso's 2017-2018 Centennial Lecture Series, El Paso, TX (October 2018)

Collegium Ramazzini Days, Carpi, Italy (November 2018)

Environment & Breast Cancer Transforming Data into Action Community Forum, Washington, DC (November 2018)

Sociedad de Toxicología de Chile Meeting, Valparaíso, Chile (November 2018)

George Washington University Seminar - Toward a Toxic-Free Supply Food Chain: Identifying Data Gaps and Opportunities for Action, Washington, DC (December 2018)

Keynote Speaker at Health Canada Science Forum, Ottawa, Canada (January 2019)

Celsius-Linnaeus Lecture and Symposium at Uppsala University, Uppsala, Sweden (February 2019)

Tribal Environmental Health Summit, Tucson, AZ (February 2019)

Arizona Disaster Research and Response Exercise, Tucson, AZ (February 2019)

Society of Toxicology Annual Meeting, Baltimore, MD (March 2019)

Federal Hearing: Senate Environment and Public Works Committee Hearing on PFAS, Washington, DC (March 2019)

NC Central University's Women's Health Awareness Conference, Durham, NC (April 2019)

Lecturer at University of North Carolina Chapel Hill, Chapel Hill, NC (April 2019)

PFAS and Other Emerging Contaminants Conference, Raleigh, NC (April 2019)

Keynote Speaker at Northeastern University's PFAS Conference, Boston, MA (June 2019)

Keynote Speaker at Unwrapped: The Health Threats of Plastics and Food Packaging Chemicals, Scotts Valley, CA (June 2019)

Water Quality Community Forum and Tour at University of Iowa, Cedar Rapids, IA (June 2019)

EHS Core Center Meeting, Cedar Rapids, Iowa (June 2019)

Triangle Global Health Consortium, Research Triangle Park, NC (October 2019)

University of Modena, Modena, Italy (October 2019)

Collegium Ramazzini (Carpi, Italy (October 2019)

House Science Committee Congressional Briefing, Washington DC (November 2019)

University of Tel Aviv, Israel (December 2019)

HERA, Barcelona (January 2020)

ANSES, Paris (February 2020)

Ichan Mt Sinai School of Medicine, NY (March 2020)

FREIA, Paris (February 2020)

Appendix A

Pittsboro, NC Town Council (October 2020)

North Carolina State University (October 2020)

Southern California University School of Public Health (November 2020)

Defend Our Health (December 2020)

Yale University School of Medicine- Pediatric Grand Rounds (January 2021)

Yale University School of Public Health (February 2021)

Wisconsin Environmental Health Network (February 2021)

Wayne State University (February 2021)

New Hampshire Safe Water Alliance (March 2021)

Chicago Center for Health and the Environment, UICC (March 2021)

State of Maine Legislature (April 2021)

Council of Scientific Society Presidents (May 2021)

University of Pittsburgh School of Public Health Commencement Address (May 2021)

Columbia River Basin Restoration Program (May 2021)

Western Washington University (June 2021)

ISCHE Fluoride Webinar (June 2021)

NAS PFAS panel (July 2021`)

EWG Conference on PFAS (July 2021)

University of Paris (November 2021)

Alaska Community Against Toxics (2021)

Michigan State University (January 2022)

Public Health Summit, Pittsburgh (February 2022)

State of Maryland Legislature (February and March 2022; February and March 2023))

State of Alaska Legislature (February 2022)

Invited Symposium/Workshop Speaker

Duke University (1982)

University of Buffalo (1983)

Rutgers University (1984)

EPA (Washington, DC, 1985)

University of Arizona (1985)

Duke VA (1986)

University of Nebraska (1986)

Appendix A

CIIT (1987)

Texas A&M University (1987)

St. John's University (1987)

Veterans Administration Medical Center, St. Louis (1987)

NTP Executive Committee (1988)

Senate Committee on Environment and Public Works (Staffers) (1989)

Health Effects Research Laboratory, Research Triangle Park, NC, 1989)

USEPA, Washington, DC (1989)

NIEHS Research Day, Research Triangle Park, NC (1989)

Duke University, Durham, NC (1990)

University of North Carolina, Chapel Hill, NC (1990)

Virginia Polytechnic Institute and State University (1990)

EOHSI; Panelist at the GLO 9 Graduation Program, Rutgers University (1991)

Cornell University (1991)

Colorado State University (1991)

Health Effects Institute (1991)

Toxicology Forum (1991)

Harvard School of Public Health (1991)

Symposium on the Health Effects of Gasoline (1991)

The Toxicology Forum (1992)

University of Connecticut (1992)

World Wildlife Fund (1992)

Genetic Toxicology Association Spring Meeting (1992)

North Carolina's Environmental Management Commission (EMC) Water Quality Committee (1992)

SEGH International Conference on Lead and Other Trace Substances (1992)

Harvard School of Public Health (1992)

University of Kansas Medical Center (1992)

Association for Governmental Toxicologist, Society for Environmental Geochemistry and Health (1992)

University of Texas (1992)

American Association for the Advancement of Science (1993)

Society of Toxicology (1993)

Air and Waste Management Association (1993)

Invited twice to NIEHS (1993)

International Congress on Health Effects of Hazardous Waste (1993)

National Conference on Dioxin (1993)

Committee to Coordinate Environmental Health and Related Programs (1993)

Appendix A

Duke University School for the Environment (1993)

Great Lakes Water Quality Board Meeting (1993)

NC State University Graduate Student Professional Development Workshop (1993)

Environmental Defense Fund (1993)

American College of Toxicology (1993)

Health Protection Branch, Health and Welfare Canada (1993)

Illinois Environmental Health Association (1993)

University of Illinois (1993)

Environmental Health Protectorate, Canada (1994); University of Pittsburgh (1994)

Chlorine Chemistry Council (1994)

NIEHS Second Annual Environmental Careers Symposium, Research Triangle Park, NC (1994)

Dioxin Reassessment Press Conference, Washington, DC (1994)

Dioxin Reassessment Press Conference, Chicago, Illinois (1994)

EPA Region 5 Dioxin Reassessment Press Conference (1994)

University of California, Davis, CA (1994)

Durham-Chapel Dietetic Association (1994)

North Carolina State University Workshop (1994)

Duke University Occupational Medicine Seminar Series (1994)

University of Kentucky, Graduate Center for Toxicology & Sigma Xi (1994)

NCSOT (1994)

National Academy of Sciences (1994)

Southern Illinois University (1994)

Air & Waste Management Association (1994)

North Carolina Bar Association (1995)

Cornell University (1995)

North American Commission for Environmental Cooperation (1995)

Tribal Council - St. Regis Mohawk Tribe Environment Division (1996)

National Wildlife Conservation (1996)

American Chemical Society (1996)

West Virginia University, Department of Pharmacology & Toxicology (1996)

University of Illinois (1996)

Cincinnati Medical Center - Institute of Environmental Health (1996)

International Symposium Dioxins and Furans, Heidelberg, Germany (1996)

University of Michigan - Consultant Program (1997)

NC State University - SCI-LINK Teachers Day, Raleigh, NC (1997)

Dosimetry for Persistent Chemicals, Washington, DC (1997)

Appendix A

University of Buffalo - Buffalo environmental health Sciences Conference, Buffalo, NY (1997)

Health Conference `97, Montreal, Canada (1997)

Dioxin `97 Symposium on Chlorinated Dioxins and Related Compounds, Indianapolis, IN (1997)

Southern Illinois University - Betram W. Carnow Memorial Symposia (1997)

50th Anniversary of the Korean Society of Pharmacology; The Dioxin Conference;
Pohang University, Seoul, Korea (1997)

Lineberger Cancer Center, University of NC at Chapel Hill (1998)

Endocrine Disruptor Workshop, Raleigh, NC (1998)

Chemical Mixtures Colloquium, Washington, DC (1998)

University of NC Research Integrity Conference (1998)

Butadiene Annual Research Review Meeting, Houston, TX (1998)

Cell Signaling Workshop, RTP, NC (1998)

Workshop on Ah Receptor-Controlled Responses in Tumor Promotion,
Germany; University of Maine, Orono, ME (1998)

Risk Characterization of Dioxin, EPA, RTP, NC (1998)

NIEHS/NTA Science Fair, RTP, NC (1998)

AMSA National Convention, Chicago, IL (1999)

Graduate Student Convocation, ASPET Meeting, Washington, DC (1999)

Washington State University, Pullman, WA (1999)

Environmental Mutagen Society (2000)

Bowman Gray School of Medicine (2000)

Endometriosis 2000, London (2000)

University of Wisconsin, Madison (2000)

Physicians for Social Responsibility, Washington, DC (2000)

Ecology and Health Conference, Raleigh, NC (2000)

Dioxin 2000, Monterey and Berkley, California (2000)

American Public Health Association Annual Meeting (2000)

University of New Mexico Toxicology Program (2000)

Cornell University (2001)

University of Zurich (2001)

Karolinska Institute (2001)

Local Motion, Detroit, MI (2001)

Michigan Department of Environmental Quality (2002)

University of Illinois, Chicago (2002)

North Carolina State University (2003)

Loma Linda University, CA (2003)

Appendix A

Boston City Council, Boston, MA (2003)

Boston City Council on Dioxin, Boston, MA (2003)

Research Triangle Park Career Evaluation, Research Triangle Park, NC (2003)

University of California at Los Angeles (2004)

Harvard Seminar, Boston, MA (2004)

EMS Panel Debate, Washington, DC (2004)

Duke University Integrated Toxicology Program, Durham, NC (2004)

Lone Tree Council, Saginaw Bay Watershed, Saginaw, MI (2005)

Porter School of Environmental Studies and Haifa University, Israel (2005)

Michigan Department of Environmental Quality, Midland, Michigan (2005)

University of Southern Maine, Portland, Maine (2005)

North Carolina State University, Dept. of Environmental and Molecular Toxicology Seminar, Raleigh, NC (2006)

Southwestern Medical University, Dallas, TX (2006)

University of Wisconsin, Madison, WI (2006)

DECA, Washington, DC (2006)

University of Michigan, Ann Arbor, Michigan (2006)

Brominated Flame Retardants (BFR), Washington, DC (2006)

Environmental Partnership Summit, Research Triangle Park, NC (2006)

232nd American Chemical Society Meeting & Exposition, San Francisco, CA (2006)

Weybridge + 10 Workshop, Helsinki, Finland (2006)

International Conference on Food Contaminants and Neurodevelopmental Disorders, Valencia, Spain (2006)

PALs Meeting, Crystal City, Arlington, VA (2006)

East Carolina University (2007)

U.S. EPA Region 8, Denver, CO (2007)

NIEHS Meeting, Research Triangle Park, NC (2007)

P.O.P. Culture, Santa Monica, CA (2007)

Evaluation of the human relevance of modes of action in animals,

University of North Carolina, Chapel Hill, North Carolina (2008)

Evaluating the human relevance of modes of action in Animals Workshop,
ILSI Research Foundation University of North Carolina, Chapel Hill, NC (2008)

Duke ITP, Duke University, Durham, North Carolina (2008)

Indiana University, Bloomington, Indiana (2008)

National Public Radio (2009)

Frontline (2009)

CNN (2009)

Summers of Discovery Seminar Series, Research Triangle Park, NC (2009)

Appendix A

Computational Toxicology Webinar Series, Research Triangle Park, NC (2009)

National Conversation on Chemical Exposures, Washington, DC (2009)

Great Lakes Green Chemistry Network Seminar, College Park, MD (February 2010)

UNC Institute of the Environment, Chapel Hill, NC (March 2010)

University of Virginia Plastic Project, Charlottesville, VA (April 2010)

University of Illinois at Chicago School of Public Health (May 2010)

Wake Forest University, Wake Forest, NC (October 2010)

James L. Whittenburg Lecture, Boston, MA (December 2010)

Cutting Edge Research on Environmental Health, Israel (February 2011)

University of Haifa, Israel (February 2011)

University of Washington School of Public Health, Seattle, WA (March 2011)

Congressman Price Science Panel, Research Triangle Park, NC (June 2011)

Los Angeles Community Forum, CA (October 2011)

Duke Integrated Toxicology Environmental Health Program, Durham, NC (November 2011)

Texas Women's University Annual Celebration of Science, Denton, TX (March 2012)

University of Montana, Missoula, MT (May 2012)

The Horizons @ Heinz, Heinz, Center, Washington, DC (May 2012)

AAAS Barnard Lecture, Washington, DC (May 2012)

University of Rochester, Toxicology Retreat, Rochester, NY (May 2012)

Eunice Kennedy Shriver National Institute of Child Health and Human Development
Washington, DC (November 2012)

University of Puerto Rico, San Juan, PR (February 2013)

Panelist on two panels at the Panels on Women's Cancers, Hosted by Fran Drescher, with Representative Deutch, Washington, DC (September 2013)

44th Annual Homer N. Calver Lecturer, APHA, Boston, MA (November 2013)

University of North Carolina Chapel Hill, School of Public Health, Chapel Hill, NC (March 2014)

Department of Molecular Biomedical Sciences Seminar Series,
North Carolina State University, Raleigh, NC (April 2014)

U.S. Environmental Protection Agency Cutting Edge Speaker Series, RTP, NC (April 2014)

Rockland County Office of Aging, Invited to Speak to the AARP Group by Representative Lowey (NY) on the Environment and NIEHS, Rockland County AARP, Rockland County, NY (April 2014)

Mailman School of Public Health, Sewell Lecture Series Guest Lecturer,
Columbia University, New York City, NY (April 2014)

Toxicology Scholars Colloquium Guest Lecturer, School of Pharmacy,
University of Connecticut; Storrs, CT (April 2014)

Ben-Gurion University of the Negev, Seminar, Beer-Sheva, Israel (May 2014)

Environment and Health Fund & Israeli Ministry of Health Meeting,
Hebrew University, Jerusalem, Israel (May 2014)

Appendix A

Meeting on Women's Health sponsored by Representative Lowey,
Mercy College, Dobbs Ferry, NY (July 2014)

North Carolina State University Distinguished Speaker Series, Raleigh, NC (September 2014)

Yale University, Environmental Health Seminar Series, New Haven, CT (December 2014)

Office of Budget Management Tour of the National Center for Advancing Translational Sciences (NCATS) Tox21 Facility, Speaker on Tox21 Accomplishments, Rockville, MD (April 2015)

Invited Lecturer, NIH Leaders Seminar Series, University of Illinois at Urbana-Champaign, IL (April 2015)

Welcome Speaker, Women's Health Awareness Day, North Carolina Central University, NC (April 2015)

Georgetown University, Washington, DC (April 2015)

Brooklyn Community Conversation on Toxics, Climate Change & Health, Brooklyn, NY (May 2015)

Elucidating Environmental Dimensions of Neurological Disorders and Diseases: Understanding New Tools from Federal Chemical Testing Programs, University of California Davis, Davis, CA (June 2015)

Weill Cornell Medical School, New York, NY (February 2016)

Icahn School of Medicine at Mount Sinai, New York, NY (March 2016)

Invited Speaker, Fogarty Scholars and Fellows Orientation, Bethesda, MD (July 2016)

Invited Speaker, Northeastern University, Boston, MA (July 2016)

Triangle Global Health Consortium Annual Conference, Chapel Hill, NC (September 2016)

Research! Louisville 2016, University of Louisville, KY (October 2016)

Virginia Tech Carilion Research Institute, Roanoke, VA (October 2016)

Global Climate Change: Interdisciplinary Perspectives,
University of North Carolina, Chapel Hill, NC (October 2016)

Friend of NIEHS 50th Anniversary Congressional Briefing, Washington, DC (November 2016)

Autism Grantee Meeting, Durham, NC (December 2016)

Environmental Health Science FEST, Durham, NC (December 2016)

Annual Friends of NIEHS Annual Meeting, Washington, DC (January 2017)

Jewish Community Center, Durham, NC (February 2017)

Center for Human Health and the Environment Science Symposium,
North Carolina State University, Raleigh, NC (February 2017)

Interagency Coordinating Committee on the Validation of Alternative Methods (ICCVAM) Workshop on Modernizing the Safety Assessment of Drugs and Chemicals, Bethesda, MD (February 2017)

Penn State Institutes for Energy and the Environment, Penn State University, University Park, PA (April 2017)

Keynote Address at Environmental Justice and the Future of Environmental Health Research, Rutgers University, New Brunswick, NJ (April 2017)

RTI Fellows Program Distinguished Lecture, Research Triangle Park, NC (May 2017)

Northeastern University Poly-Fluoroalkyl Substances (PFAS) Conference, Boston, MA (June 2017)

Fogarty Global Health Fellows Program Orientation, Bethesda, MD (July 2017)

Environmental Mutagenesis and Genomics Society Annual Meeting, Raleigh, NC (September 2017)

University of Buffalo, Buffalo, NY (September 2017)

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Colorado State University, Fort Collins, CO (September 2017)

Triangle Global Health Consortium Annual Conference, Durham, NC (September 2017)

Michael J Fox Foundation, Washington, DC (January 2018)

Friends of NIEHS Annual Meeting, Washington, DC (January 2018)

Winter Toxicology Forum, Washington, DC (January 2018)

Triangle Global Health Consortium Career Day, Durham, NC (February 2018)

All Federal Coordination of PFAS, Bethesda, MD (February 2018)

NASEM Workshop: Informing Environmental Health Decisions through Data Integration, Washington, DC (February 2018)

NIEHS Superfund Congressional Briefing, Washington, DC (March 2018)

Friends of NIEHS Congressional Briefing on Neurological Disease, Washington, DC (March 2018)

Understanding the Combined Effects of Environmental Chemical and Non-Chemical Stressors: Atherosclerosis as a Model Workshop, Research Triangle Park, NC (April 2018)

Women's Health Awareness Day, Durham, NC (April 2018)

Earth Day Science and Music Event, Durham, NC (April 2018)

NIEHS Career Symposium, Research Triangle Park, NC (May 2018)

Free Radicals: Past, Present, and Future, Research Triangle Park, NC (May 2018)

NC Women of Color Research Network First Annual Spring Conference, Research Triangle Park, NC (May 2018)

NC State and NIEHS Summer Research Program, Research Triangle Park, NC (June 2018)

Congressional Briefing with HHS Deputy Secretary and NIH on NTP Systematic Review of Monograph on Sarin, Cell Phone Radiofrequency Radiation, CLAIRTY_BPA, Report on Carcinogens 15th Edition, Washington, DC (July 2018)

PFAS Congressional Briefings with the Office of U.S. Senator Gary Peters of Michigan and the Office of U.S. Senator Jeanne Shaheen of New Hampshire, Washington, DC (July 2018)

Assembly of Scientists Summer Meeting, Research Triangle Park, NC (August 2018)

Developing a Data Science Competent EHS Workforce Workshop, Research Triangle Park, NC (August 2018)

Swedish Toxicology Sciences Research Center (SWETOX) Academy Workshop, Stockholm, Sweden (August 2018)

Science and Policy of Organohalogens Workshop, Ottawa, Canada (August 2018)

Metabolomics Common Fund Kick Off, Research Triangle Park, NC (September 2018)

NTP Workshop on Circulating, Cell-free DNA as a Strategy to Identify Novel Biomarkers and Mediators of Inflammation in Environmental Exposures and Disease, Research Triangle Park, NC (September 2018)

Environmental Epidemiology Workshop w/Health and Environmental Sciences Institute, Research Triangle Park NC (October 2018)

Friends of NIEHS Congressional Briefing for Child Health Month, Washington, DC (October 2018)

ORWH Pearls of Wisdom Video Interview, Washington, DC (October 2018)

NC Scholars Connect Program Seminar, Research Triangle Park, NC (October 2018)

NIEHS and EPA Children's Center Meeting, Research Triangle, Park, NC (October 2018)

Assembly of Scientists Winter Meeting, Research Triangle Park, NC (December 2018)

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National Academy of Sciences Workshop: ESEHS Workshop 1 - Understanding the Interplay of Environmental Stressors, Infectious Disease, and Human Health, Washington, DC (January 2019)

Friends of NIEHS Annual Meeting, Washington, DC (January 2019)

NC Public Health Leaders Conference, Raleigh, NC (January 2019)

Triangle Global Health Consortium Career Day, Research Triangle Park, NC (February 2019)

North Carolina State University's CHHE 3rd Annual Symposium- Exploring PFAS in North Carolina: Impacts on the Environment and Human Health, Raleigh, NC (February 2019)

Four Corners Interior Congressional Briefing, Washington, DC (February 2019)

National Academy of Sciences Workshop: ESEHS Workshop 2: The Promise of Single Cell and Single Molecule Analysis Tools to Advance Environmental Health Research (March 2019)

International Agency for Research on Cancer – International Women's Day Symposium, Lyon, France (March 2019)

Third International Workshop on Chronic Kidney Disease of Unknown Origin (CKDu) in Mesoamerica and Other Regions, San José, Costa Rica (March 2019)

Trans-NIH Workshop on Inflammation Resolution Biology, Research Triangle Park, NC (March 2019)

National Trainees Assembly Spring GA Meeting, Research Triangle Park, NC (April 2019)

Video Interview: European TV Channel "Arte", Bethesda, MD (April 2019)

Congressional Briefing: Senate Environment and Public Works Committee Majority Staff on PFAS, Washington, DC (April 2019)

Congressional Briefing: FY 20 NIEHS Superfund-related activities briefing for House and Senate Interior & Environment Appropriations Subcommittee Staff, Washington, DC (April 2019)

NIEHS Career Symposium, Research Triangle Park, NC (April 2019)

NTP Workshop: Converging on Cancer, Washington, DC (April 2019)

Annual Symposium of the Society of Toxicologic Pathology, Raleigh, NC (June 2019)

Deichman Lecture, 16th International Congress on Toxicology, Honolulu, Hawaii (July 2019)

ICCVAM (October 2019)

Environmental Health Fund, Jerusalem, Israel (December 2019)

HERA Annual Meeting, Barcelona, Spain (January 2020)

ANSES, Paris, France (February 2020)

EURION Annual Meeting, Paris, France (February 2020)

University of Michigan "From PBBs to PFAS", Ann Arbor, Michigan (February 2020)

Icahn Mt. Sinai School of Medicine Exposome Symposium (March 2020)

Virtual Six Classes Retreat (May 2020)

Virtual 20th Anniversary of ICCVAM (May 2020)

Virtual Society of Birth Defects and Prevention (June 2020) -Josef Warkany Lecture

Virtual Symposium, Skaggs School of Pharmacy, University of Colorado (August 2020)

UCSF PRHE Science Response Network: Setting a new scientific agenda for chemical policy (September 2020)

URI STEER: PFAS In Our World (October 2020)

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Yale School of Public Health (October 2020)

Toxic Free Future for our Children (December 2020)

BizNGO2020 (December 2020)

American Geophysical Union (December 2020)

University of Cincinnati Center for Environmental Genetics Research Symposium (March 2021)

Society of Toxicology Workshop on Flame Retardants (March 2021)

Columbia River Basin Restoration Program (May 2021)

ISCHE Fluoride Webinar (June 2021)

NAS PFAS panel (July 2021`)

EWG Conference on PFAS (July 2021)

Chicago Waterworks – Argonne and EPA Region5, Keynote (October 2021)

INSERM-Sorbonne-University of Paris, History of Environmental Public Health Keynote (November 2021)

Gil Omenn and Margaret Darling Environmental Health Inaugural Lecture, University of Washington (December 2021)

Michigan State University (January 2022)

PPToxVII Keynote (January 2022)

Environmental Health Project Public Health Summit (February 2022)

Society of Toxicology Merit Award Lecture (March 2022)

Yale Winslow Award Lecture (April, 2022)

PFAS Disposal Symposium (May 2022)

Vietnam Veterans of America (August 2022)

NIEHS Breast Cancer Symposium (August 2023)

University of Arizona Global Health Symposium (September 2022)

Dioxin2023, International Symposium on POPs, Plenary (October 2023)

Duke Global Health Symposium (October 2022)

AAAS Seminar on PFAS and CERCLA (October 2022)

University of Southern California John Peters Memorial Lecture (November 2022)

Breast Cancer and Silent Spring (November 2022)

Duke Integrated Toxicology Program (January 2023)

CHE Café (February 2023)

Minnesota Legislature (March 2023)

University of California, Davis (April 2023)

Appendix A

Major Committee Responsibilities

NIEHS Radiation Safety Committee (1985-1989)

Mouse Strains for Carcinogenesis Studies (1985)

NIEHS Research Support Subcommittee (1986)

NIEHS Laboratory Casework Committee (1986-1989)

Judge, SOT Mechanism Section Graduate Student Awards (1985, 1986) Treasurer, NC SOT (1986-1988)

TRTP Promotion Committee (1984-1989), Chairman (1987-1989)

Vice President, NC SOT (1988-1989); President (1989-1990)

NAS Committee on Chemical Toxicity and Aging (1986-1988)

U.S. EPA Science Advisory Board - Halogenated Solvents Subcommittee (1987-1989)

N.J. EPA Science Advisory Board (1987-1989)

SOT Mechanism Section, Nominations Committee (1987-1989)

WHO, IPCS, Co-chair - Aging and Toxicity (1988-1993)

US EPA, Science Advisory Board - Dioxin Review (1988-1989)

DTRT, Ad Hoc Group on Future Research Priorities, Facilitator (1988-1989)

Education Committee, SOT (1989-1992)

NIOSH Peer Review Board on Dioxin Studies (1989-1994)

CIIT, Dioxin Review Panel (1990-1994); Scientific Advisory Panel (1990)

USEPA/ORD - Committee on Scientific Ethics (1990-1995)

ILSI Committee on Pharmacokinetics (1992-1996)

SOT, Nominating Committee (1993-1994)

SOT, Mechanisms Section (V.P. 1992-1993; Pres. 1993-1994)

HERL Symposium Committee (1992-present)

U.S. EPA Laboratory Implementation Committee, Science and Scientific Subcommittee,

Co-Chair of the Scientific and Scientific Career Subcommittee – Fellowship Committee Chair

Fellowship Committee (1994-1999)

Member - ORD Human Resources Committee (1999)

Member of the Chemical Manufacturers Association Butadiene Panel (1992-1996)

External Advisory Committee for the NIEHS Planning Grant for an EHS Center (1993-1995)

Executive Committee of the Division of Toxicology, ASPET (1994-1997)

SOT Council (1996-1999)

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ORD/OW Arsenic External RFA Committee (1996-1998)

Executive Committee, International Society for the Study of Xenobiotics (1996-1999)

International Organizing Committee, Dioxin (1992-2007)

Awards Committee, SOT (1998-2000)

Liaison - NERL/NHEERL Interaction Workgroup (1998-2007)

Chair, Division of Toxicology, ASPET (1998-2000)

Executive Committee of the RTP Drug Metabolism Discussion Group (2003)

Agency Wide PBT Initiative (1998-2005)

Society of Toxicology – Presidential Chain (2002-2006)

HESI Biomonitoring Technical Committee (2006)

Institute of Medicine Roundtable on Public Health and the Environment (2010-2018)

National Academy of Science, Medicine, and Engineering: Emerging Environmental Health Issues (2018-2021)

Member of Scientific and Policy Advisory Board Global PFAS Science Panel, Swiss Federal Institute of Technology, Zurich Institute of Biogeochemistry and Pollutant Dynamics (2018)

Scientific Advisory Board, FREIA (2019-2023)

International Advisory Board, EURION (2019-2023)

International Advisory Board, HERA (2019-2023)

National Academy of Medicine: Standing committee to Advise the Department of State on Unexplained Health Effects on US Government Employees and their Families at Overseas Embassies (2019-2020)

Sloan Foundation Advisory Board on Indoor Air Contaminants (2020-2023)

National Academy of Medicine Workshop on Companion Animals as Sentinels for Environmental Exposure (2020-2022) – Chair

Science Advisory Board, EaRTH Center, UCSF (2020-2024)

Veterans Administration Air Force Health Study Committee Chair (2021-2025)

Appendix A

Research Advisor for

Dennis Darcey, MS, University of North Carolina, Department of Environmental Sciences and Engineering, 1982

Susan Borghoff, MS, Ph.D., University of North Carolina, Department of Environmental Sciences and Engineering, 1987

Chris Miller, MS, University of North Carolina, Department of Environmental Sciences and Engineering, 1985

Charles Hebert, Ph.D., University of North Carolina, Toxicology Curriculum, 1990

Laurie Couture-Haws, MS, Ph.D., University of North Carolina, Department of Environmental Sciences and Engineering and Toxicology Curriculum (MS 1987; Ph.D. 1990)

Yolanda Banks Anderson, Ph.D., University of North Carolina, Department of Environmental Sciences and Engineering, 1990

Lorrene Kedderis, Ph.D., University of North Carolina, Toxicology Curriculum, 1992

Mary K. McKinley, MS, Duke University, School of the Environment, 1992

Renu Batra, Ph.D., University of North Carolina, Department of Environmental Sciences and Engineering (deceased)

Krista Little Johnson, MS, University of North Carolina, Department of Environmental Sciences and Engineering, 1996

Christopher Hurst, Ph.D., University of North Carolina, Curriculum in Toxicology, 1999

Deborah Burgin, Ph.D., University of North Carolina, Curriculum in Toxicology, 2005

Daniele Staskal, Ph.D., University of North Carolina, Curriculum in Toxicology, 2005

Daniel Bauer, Ph.D., University of North Carolina, Curriculum in Toxicology, withdrew

Michele La Merrill, Ph.D., University of North Carolina, Curriculum in Toxicology, 2008

David Szabo, Ph.D., University of North Carolina, Curriculum in Toxicology, 2011

Alicia Richards, MPH, University of North Carolina, Department of Environmental Sciences and Engineering, 2018

Thesis Committee (Ph.D. Students)

Alan Jo Cato, University of North Carolina, School of Pharmacy

Charlie Sewall, University of North Carolina, Toxicology Curriculum

Joost DeJongh, University of Utrecht, The Netherlands

Angelique Van Birgelen, University of Utrecht, The Netherlands

George Monteverdi, Duke University, School of the Environment

Chia-Yang Chen, University of North Carolina, School of Public Health

Coralie Groenveld, Agricultural University of Wageningen, Wageningen, The Netherlands

Irene Kampen, Agricultural University of Wageningen, Wageningen, The Netherlands

Michael Wyde, UNC, Curriculum in Toxicology

Jie (Jane) Dong, Duke University, School of the Environment

Yo Chan Jeong, University of North Carolina, School of Public Health, 2005

Lieke Peters, University of Utrecht, The Netherlands, 2006

Oliver Hamblett, Harvard School of Public Health

Pamela Noyes, Duke School of the Environment

Thuy Lam, Harvard School of Medicine

Samantha Van Etten, University of Buffalo, 2021

Liora Fiksel, MPH, Yale University, 2022

Appendix A

Tess Leuthner, Duke, 2022-23

Postdoc Advisor

L.R. (Mark) Kao, 1984-1985

Dave Brewster, 1985-1987

Usha Gundimeda, 1986-1988

Barbara Abbott, 1987-1989

Tim McMahon, 1988-1990

Mike DeVito, 1991-1994

Angelique Van Birgelen, 1994-1997

Xiaofeng Wang, 1995-1997

Michael Santostefano, 1994-1999

Brian Slezak, 1997-1999

Jonathan Hamm, 1997-2000

Claude Emond, 2001-2004

Lisa Vinikoor, 2008-2009

Sally White, 2008-2009

Gabriel Knudsen, 2011-2014

Journal Articles

1. Page J, Whaley P, Bellingham M, Birnbaum LS, Cavoski A, Fetherston Dilke D, Garside R, Harrad S, Kelly F, Kortenkamp A, Martin O, Stec A, Woolley T. A new consensus on reconciling fire safety with environmental & health impacts of chemical flame retardants. Environment international. 2023:107782. doi: https://doi.org/10.1016/j.envint.2023.107782.

2. Woodruff TJ, Rayasam SDG, Axelrad DA, Koman PD, Chartres N, Bennett DH, Birnbaum LS, Brown P, Carignan CC, Cooper C, Cranor CF, Diamond ML, Franjevic S, Gartner EC, Hattis D, Hauser R, Heiger-Bernays W, Joglekar R, Lam J, Levy JI, MacRoy PM, Maffini MV, Marquez EC, Morello-Frosch R, Nachman KE, Nielsen GH, Oksas C, Abrahamsson DP, Patisaul HB, Patton S, Robinson JF, Rodgers KM, Rossi MS, Rudel RA, Sass JB, Sathyanarayana S, Schettler T, Shaffer RM, Shamasunder B, Shepard PM, Shrader-Frechette K, Solomon GM, Subra WA, Vandenberg LN, Varshavsky JR, White RF, Zarker K, Zeise L. A science-based agenda for health-protective chemical assessments and decisions: overview and consensus statement. Environ Health. 2023;21(Suppl 1):132. Epub 2023/01/13. doi: https://doi.org/10.1186/s12940-022-00930-3. PubMed PMID: 36635734.

3. Maffini MV, Rayasam SDG, Axelrad DA, Birnbaum LS, Cooper C, Franjevic S, MacRoy PM, Nachman KE, Patisaul HB, Rodgers KM, Rossi MS, Schettler T, Solomon GM, Woodruff TJ. Advancing the science on chemical classes. Environ Health. 2023;21(1):120. doi: https://doi.org/10.1186/s12940-022-00919-y. PubMed PMID: 36635752.

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4. Ben Ishai P, Davis D, Taylor H, Birnbaum L. Problems in evaluating the health impacts of radio frequency radiation. Environmental research. 2023:115038. doi: https://doi.org/10.1016/j.envres.2022.115038.

5. Veneri F, Vinceti M, Generali L, Giannone ME, Mazzoleni E, Birnbaum LS, Consolo U, Filippini T. Fluoride exposure and cognitive neurodevelopment: Systematic review and dose-response meta-analysis. Environmental research. 2023:115239. doi: https://doi.org/10.1016/j.envres.2023.115239.

6. Wikoff D, Ring C, DeVito M, Walker N, Birnbaum L, Haws L. Development and Application of a Systematic and Quantitative Weighting Framework to Evaluate the Quality and Relevance of Relative Potency Estimates for Dioxin-Like Compounds (DLCs) for Human Health Risk Assessment. Regulatory Toxicology and Pharmacology. 2023;Submitted.

7. Birnbaum L. Environmental Health: Past, Present, and Future. Comité pour l'histoire de l'Inserm. 2022;2/2(4):72-6. doi: http://hdl.handle.net/10608/12454.

8. Kay JE, Cardona B, Rudel RA, Vandenberg LN, Soto AM, Christiansen S, Birnbaum LS, Fenton SE. Chemical Effects on Breast Development, Function, and Cancer Risk: Existing Knowledge and New Opportunities. Current environmental health reports. 2022. Epub 2022/08/20. doi: https://doi.org/10.1007/s40572-022-00376-2. PubMed PMID: 35984634.

9. Chartres N, Sass JB, Gee D, Bălan SA, Birnbaum L, Cogliano VJ, Cooper C, Fedinick KP, Harrison RM, Kolossa-Gehring M, Mandrioli D, Mitchell MA, Norris SL, Portier CJ, Straif K, Vermeire T. Conducting evaluations of evidence that are transparent, timely and can lead to health-protective actions. Environmental health : a global access science source. 2022;21(1):123. Epub 2022/12/06. doi: https://doi.org/10.1186/s12940-022-00926-z. PubMed PMID: 36471342.

10. Rider CV, Birnbaum LS, DeVito MJ, Hertzberg RC, Rice GE, Teuschler LK. In Memoriam: Jane Ellen Simmons. Environmental health perspectives. 2022;130(10):101601. Epub 2022/10/28. doi: https://doi.org/10.1289/ehp12225. PubMed PMID: 36300649; PMCID: PMC9608555.

11. Cave MC, Pinkston CM, Rai SN, Wahlang B, Pavuk M, Head KZ, Carswell GK, Nelson GM, Klinge CM, Bell DA, Birnbaum LS, Chorley BN. Circulating MicroRNAs, Polychlorinated Biphenyls, and Environmental Liver Disease in the Anniston Community Health Survey. Environmental health perspectives. 2022;130(1):17003. Epub 2022/01/07. doi: https://doi.org/10.1289/ehp9467. PubMed PMID: 34989596; PMCID: PMC8734566.

12. Petriello MC, Mottaleb MA, Serio TC, Balyan B, Cave MC, Pavuk M, Birnbaum LS, Morris AJ. Serum concentrations of legacy and emerging per- and polyfluoroalkyl substances in the Anniston Community Health Surveys (ACHS I and ACHS II). Environment international. 2022;158:106907. Epub 2021/11/12. doi: https://doi.org/10.1016/j.envint.2021.106907. PubMed PMID: 34763231; PMCID: PMC9131314.

13. Dinse GE, Co CA, Parks CG, Weinberg CR, Xie G, Chan EKL, Birnbaum LS, Miller FW. Expanded assessment of xenobiotic associations with antinuclear antibodies in the United States, 1988-2012. Environment international. 2022;166:107376. Epub 2022/07/06. doi: https://doi.org/10.1016/j.envint.2022.107376. PubMed PMID: 35785669.

Appendix A

14. Birnbaum LS. Op-Ed: FDA fails to protect the public from chemicals health risks. Environmental Health News. 2022. doi: https://www.ehn.org/fda-chemical-regulation-2657184101/fdas-2013-review-a-commitment-unfulfilled.

15. Salvatore D, Mok K, Garrett KK, Poudrier G, Brown P, Birnbaum LS, Goldenman G, Miller MF, Patton S, Poehlein M, Varshavsky J, Cordner A. Presumptive Contamination: A New Approach to PFAS Contamination Based on Likely Sources. Environ Sci Technol Lett. 2022. doi: https://doi.org/10.1021/acs.estlett.2c00502.

16. Southerland E, Sussman R, Birnbaum L. Unjustified industry pushback on EPA's toxic chemical regulation. The Hill. 2022. doi: https://thehill.com/opinion/energy-environment/3495942-unjustified-industry-pushback-on-epas-toxic-chemical-regulation/.

17. Peecher JS, Stromberg A, Lu H, Quynh HT, Schecte AJ, Weng J, Crandall R, Birnbaum LS. Biomonitoring of Polybrominated Dioxins & Furans, Polychlorinated Dioxins & Furans, and Dioxin Like Polychlorinated Biphenyls in Vietnamese Female Electronic Waste Recyclers. Journal of occupational and environmental medicine. 2022;64(9):742-7. Epub 2022/02/06. doi: https://doi.org/10.1097/JOM.0000000000002506. PubMed PMID: 35121692; PMCID: PMC9680905.

18. Birnbaum LS, Taylor HS, Baldwin H, Ben-Ishai P, Davis D. RE: Cellular Telephone Use and the Risk of Brain Tumors: Update of the UK Million Women Study. Journal of the National Cancer Institute. 2022;114(11):1551-2. doi: https://doi.org/10.1093/jnci/djac110.

19. Post GB, Birnbaum LS, DeWitt JC, Goeden H, Heiger-Bernays WJ, Schlezinger JJ. Letter to the editors regarding "The conundrum of the PFOA human half-life, an international collaboration.". Regulatory Toxicology and Pharmacology. 2022. doi: https://doi.org/10.1016/j.yrtph.2022.105240.

20. Birnbaum LS, Bornehag CG. Phthalates Should Be Regulated as a Class to Protect the Brains of Our Children. American journal of public health. 2021;111(4):551-2. Epub 2021/03/11. doi: https://doi.org/10.2105/ajph.2021.306193. PubMed PMID: 33689442; PMCID: PMC7958052

21. Patisaul HB, Behl M, Birnbaum LS, Blum A, Diamond ML, Rojello Fernández S, Hogberg HT, Kwiatkowski CF, Page JD, Soehl A, Stapleton HM. Beyond Cholinesterase Inhibition: Developmental Neurotoxicity of Organophosphate Ester Flame Retardants and Plasticizers. Environmental health perspectives. 2021;129(10):105001. Epub 2021/10/07. doi: https://doi.org/10.1289/ehp9285. PubMed PMID: 34612677; PMCID: PMC8493874.

22. Barouki R, Kogevinas M, Audouze K, Belesova K, Bergman A, Birnbaum L, Boekhold S, Denys S, Desseille C, Drakvik E, Frumkin H, Garric J, Destoumieux-Garzon D, Haines A, Huss A, Jensen G, Karakitsios S, Klanova J, Koskela IM, Laden F, Marano F, Franziska Matthies-Wiesler E, Morris G, Nowacki J, Paloniemi R, Pearce N, Peters A, Rekola A, Sarigiannis D, Šebková K, Slama R, Staatsen B, Tonne C, Vermeulen R, Vineis P. The COVID-19 pandemic and global environmental change: Emerging research needs. Environment international. 2021;146:106272. Epub 2020/11/26. doi: https://doi.org/10.1016/j.envint.2020.106272. PubMed PMID: 33238229; PMCID: PMC7674147.

23. Cordner A, Goldenman G, Birnbaum LS, Brown P, Miller MF, Mueller R, Patton S, Salvatore DH, Trasande L. Correction to The True Cost of PFAS and the Benefits of Acting Now. Environmental science

& technology. 2021. Epub 2021/09/03. doi: https://doi.org/10.1021/acs.est.1c04938. PubMed PMID: 34472851.

24. Cordner A, Goldenman G, Birnbaum LS, Brown P, Miller MF, Mueller R, Patton S, Salvatore DH, Trasande L. The True Cost of PFAS and the Benefits of Acting Now. Environmental science & technology. 2021;55(14):9630-3. Epub 2021/07/08. doi: https://doi.org/10.1021/acs.est.1c03565. PubMed PMID: 34231362; PMCID: PMC8296683.

25. Kwiatkowski CF, Andrews DQ, Birnbaum LS, Bruton TA, DeWitt JC, Knappe DRU, Maffini MV, Miller MF, Pelch KE, Reade A, Soehl A, Trier X, Venier M, Wagner CC, Wang ZY, Blum A. Response to "Comment on Scientific Basis for Managing PFAS as a Chemical Class". Environ Sci Technol Lett. 2021;8(2):195-7. doi: https://doi.org/10.1021/acs.estlett.1c00049.

26. Karlsson O, Rocklöv J, Lehoux AP, Bergquist J, Rutgersson A, Blunt MJ, Birnbaum LS. The human exposome and health in the Anthropocene. Int J Epidemiol. 2021;50(2):378-89. Epub 2020/12/23. doi: https://doi.org/10.1093/ije/dyaa231. PubMed PMID: 33349868; PMCID: PMC8128460.

27. Emond C, DeVito MJ, Birnbaum LS. A PBPK model describing the pharmacokinetics of γ-HBCD exposure in mice. Toxicology and applied pharmacology. 2021;428:115678. Epub 2021/08/15. doi: https://doi.org/10.1016/j.taap.2021.115678. PubMed PMID: 34390738; PMCID: PMC8674938.

28. Castro L, Liu J, Yu L, Burwell AD, Saddler TO, Santiago LA, Xue W, Foley JF, Staup M, Flagler ND, Shi M, Birnbaum LS, Dixon D. Differential Receptor Tyrosine Kinase Phosphorylation in the Uterus of Rats Following Developmental Exposure to Tetrabromobisphenol A. Toxicol Res Appl. 2021;5. Epub 2022/01/25. doi: https://doi.org/10.1177/23978473211047164. PubMed PMID: 35071781; PMCID: PMC8774279.

29. Knudsen TB, Fitzpatrick SC, De Abrew KN, Birnbaum LS, Chappelle A, Daston GP, Dolinoy DC, Elder A, Euling S, Faustman EM, Fedinick KP, Franzosa JA, Haggard DE, Haws L, Kleinstreuer NC, Buck Louis GM, Mendrick DL, Rudel R, Saili KS, Schug TT, Tanguay RL, Turley AE, Wetmore BA, White KW, Zurlinden TJ. FutureTox IV Workshop Summary: Predictive Toxicology for Healthy Children. Toxicological sciences : an official journal of the Society of Toxicology. 2021;180(2):198-211. Epub 2021/02/09. doi: https://doi.org/10.1093/toxsci/kfab013. PubMed PMID: 33555348; PMCID: PMC8041457.

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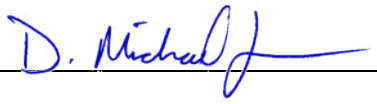
Exhibit B

D. Michael Johns, Ph.D., Relative Risks of OU2 Contaminants of Concern & Comparison with OU4

A summary of data information regarding relative ecological risks for the Lower Passaic River

March 22, 2023

Report by Dr. D. Michael Johns 

Prepared by: Windward environmental LLC

200 First Avenue West, Suite 500 ♦ Seattle, Washington ♦ 98119

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Acronyms

BERA	baseline ecological risk assessment
CBR	critical body residue
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
DDD	dichlorodiphenyldichloroethane
DDE	dichlorodiphenyldichloroethylene
DDT	dichlorodiphenyltrichloroethane
COC	contaminant of concern
COPEC	contaminant of potential ecological concern
EF	extrapolation factor
EPC	exposure point concentration
ERA	ecological risk assessment
FFS	focused feasibility study
HPAH	high-molecular-weight polycyclic aromatic hydrocarbon
HQ	hazard quotient
LOAEL	lowest-observed-adverse-effect limit
LOE	line of evidence
LPAH	low-molecular-weight polycyclic aromatic hydrocarbon
LPR	Lower Passaic River
LPRSA	Lower Passaic River Study Area
NBSA	Newark Bay Study Area
NOAEL	no-observed-adverse-effect limit
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
OU	Operable Unit
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
RI/FS	remedial investigation/feasibility study
RM	river mile
ROD	Record of Decision
TCDD	tetrachlorodibenzo-*p*-dioxin
TEQ	toxicity equivalent

total DDx	sum of all six DDT-related compounds (2,4′-DDD, 4,4′-DDD, 2,4′-DDE, 4,4′-DDE, 2,4′-DDT and 4,4′-DDT)
TRV	toxicity reference value
TSI	Tierra Solutions Inc.
USEPA	US Environmental Protection Agency
Windward	Windward Environmental LLC

1 D. Michael Johns, PhD Qualifications

I am an environmental consultant at Windward Environmental LLC (Windward) and hold a PhD in oceanography with an emphasis in marine biology from the Belle W. Baruch Institute, University of South Carolina. Over my career, I have focused on the assessment and remediation of environmental contamination under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and analogous state programs. My principal area of expertise is identifying and assessing contaminants in aquatic environments to determine any associated risks and the need for remediation.

My experience comprises 43 years of professional work at aquatic sites located throughout the United States. The varied sites where I have had the opportunity to work have provided me with a broad knowledge base on issues pertaining to the assessment and remediation of contaminants. Prior to consulting, I served as a Principal Investigator at the U.S. Environmental Protection Agency (USEPA) national research laboratory in Narragansett, Rhode Island. During my tenure there, I was a member of the team developing the datasets required to set water quality criteria for metals. I also researched methods for evaluating sublethal effects on aquatic invertebrates from contaminant exposure, and I was a Lead Investigator (1980–1985) on the Field Verification Program. This program was a joint project between the USEPA laboratory and the US Army Corps of Engineers Waterways Experiment Station to evaluate the impacts of disposing of contaminated dredged material using multiple disposal techniques. Under the Field Verification Program, I investigated lethal and sublethal impacts on benthic invertebrates using both laboratory studies and field surveys.

Recent management positions I have held as part of major aquatic contaminant assessment projects include Project Manager, Principal-In-Charge, and Senior Technical Advisor. Examples of recent, complex contaminated sites for which I have had significant technical and managerial responsibility include the Diamond Alkali Superfund site (Newark, New Jersey), Lower Duwamish River Superfund site (Seattle, Washington), and the Portland Harbor Superfund site (Portland, Oregon). Among other duties, I have been responsible for conducting and managing risk assessments, ensuring that the work performed met the requirements laid out in the administrative order for the site and was consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP).

2 Scope of Review

I have been retained by Preti Flaherty, which is representing the Small Parties Group with respect to the Lower Passaic River Study Area (LPRSA) portion of the Diamond Alkali Superfund site. I have been asked to review the Allocation Recommendation Report (AlterEcho 2020) and its underlying ecological risk methodology, and to consider whether the ecological risks associated with Operable Unit 2 (OU2) are similar to those associated with Operable Unit (OU4) such that it is reasonable to use the same risk-based methodology.

The conclusions that I present in this report are based on my review of the information and data in existence and available to EPA prior to the lodging of the settlement on December 16, 2022, including site data and studies, in addition to my own education, experience, and expertise.

3 Overview

The Lower Passaic River (LPR) is part of the Diamond Alkali Superfund site. In 1983, high levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD)[1] were detected at the Lister Avenue site. Based on these findings, in September 1984, USEPA added the Diamond Alkali Superfund site to the National Priorities List. In 1994, Occidental Chemical Corporation investigated a 6-mile stretch of the LPR (river mile [RM] 1 to RM 7) under USEPA oversight.[2] This investigation indicated that contaminated sediments moved upstream and downstream of this 6-mile stretch, leading USEPA, in 2002, to expand the investigation to include a 17.4-mile stretch of the LPR and Newark Bay as additional OUs. The Diamond Alkali Superfund site comprises four OUs:

- OU1: The first operable unit addressed, through an interim remedy, contaminated soils, groundwater, and materials at the former Diamond Alkali manufacturing facilities at 80 and 120 Lister Avenue in Newark, New Jersey, located at approximately RM 3.4 of the LPR. An interim cleanup was completed in 2001.
- OU2: The second operable unit addresses the contaminated sediments found in the lower 8.3 miles of the LPR (also referred to as the Focused Feasibility Study [FFS] Area). The nature and extent of the contamination in the lower 8.3 miles of the LPR and the associated remedial alternatives are described in two documents: the *Remedial Investigation for the Focused Feasibility Study Report for the Lower Eight Miles of the Lower Passaic River* (Louis Berger et al. 2014b) and the *Focused Feasibility Study Report for the Lower Eight Miles of the Lower Passaic River* (herein referred to as the FFS Report) (Louis Berger et al. 2014a). The Record of Decision (ROD) for OU2 was issued on March 3, 2016 (USEPA 2016).
- OU3: The third operable unit addresses the Newark Bay Study Area (NBSA), the BERA for which was completed in 2020 (Arcadis 2020). A memorandum describing the remedial action objectives and preliminary remedial goals (PRGs) for the NBSA was finalized in 2022 (Arcadis 2022).
- OU4: The fourth operable unit addresses the entire 17.4 miles of the LPRSA (including OU2). The final baseline human health risk assessment for OU4 was completed in 2017 (AECOM 2017) and the BERA was completed in 2019 (Windward 2019).

An Allocation Recommendation Report was completed in 2020 (AlterEcho 2020). The Allocation was conducted by AlterEcho. The allocation process included calculation of

[1] TCDD refers to the group of 17 dioxin and furan congeners that are evaluated as 2,3,7,8-TCDD toxicity equivalents (TEQs), or TCDD-TEQ. TCDD-TEQ is driven by the releases of 2,3,7,8-TCDD, which is the most toxic and most dominant of the 17 congeners.

[2] Occidental Chemical Corporation performed the investigation through Chemical Land Holdings, Tierra Solutions Inc. (TSI), and Maxus Energy Corporation.

the relative risk number for each COC using the eight COC's average non-cancer, cancer, and ecological harm based on the potential for risk supported by the FFS Report (Louis Berger et al. 2014a).

For this report, I reviewed the methodologies used in the OU2 BERA (Louis Berger et al. 2014a, Appendix D) and assessed their consistency with the NCP and USEPA guidance for conducting ERAs. I also reviewed the Allocation Recommendation Report (AlterEcho 2020). My conclusions on the findings of the Allocation Recommendation Report are restricted to the *Methodology for Determination of the Relative Risk of OU2 Contaminants of Concern for the Purpose of the Allocation* (Attachment A to the Allocation Protocol, which in turn is Attachment H of the Allocation Recommendation Report). Finally, I considered whether the risk-based allocation methodology used for the Allocation and the associated ecological risk could reasonably be applied and are similar to those for OU4.

4 Discussion

4.1 CONCLUSION 1: THE OU2 BERA IS CONSISTENT WITH USEPA ERA GUIDANCE.

The exposure and effects characterization assumptions and approaches, as well as risk assessment methodologies for the OU2 BERA, are consistent with USEPA's 8-step ERA guidance process (USEPA 1997), which is the standard risk assessment industry practice. Similar methodologies and lines of evidence were also evaluated for the 17-mile LPRSA (OU4) BERA (Windward 2019) and NBSA (OU3) BERA (Arcadis 2020) to determine risk estimates for ecological receptors.

- The elements of a screening-level assessment (ERA guidance Steps 1 and 2) were completed to identify contaminants of potential ecological concern (COPECs) for the OU2 BERA (FFS Report, Appendix D, Attachment 2) (Louis Berger et al. 2014a). These elements included deriving screening-level hazard quotients (HQs) based on screening-level effects and exposure concentrations.
- The elements of a baseline assessment were completed in the OU2 BERA (ERA guidance Steps 3 through 7), starting with baseline problem formulation and ending with risk characterization that included an uncertainty analysis (Louis Berger et al. 2014a, Appendix D).
- Risks were quantitively estimated in the OU2 BERA based on the use of an HQ approach comparing exposure and effect data as stated: "Individual risk estimates for a given receptor for each chemical were calculated as an HQ, which is the ratio of the EPC to a given toxicological benchmark" (FFS Report, Appendix D, Section 4.4) (Louis Berger et al. 2014a).

The OU2 BERA's (Louis Berger et al. 2014a, Appendix D) evaluation of the exposure pathways and assessment endpoints and its use of site-specific data are consistent with the NCP (40 CFR § 300.340 (d)(4)). Specific elements of the OU2 BERA consistent with ERA guidance are described in the remainder of this section.

4.1.1 The OU2 BERA used an appropriate list of COPECs

- The OU2 BERA included a screening assessment for sediment and tissue COPECs based on the Pathway Analysis Report (Battelle 2005), wherein maximum site sediment concentrations were compared to screening levels based on published compilations; it also considered a bioaccumulation screen (i.e., tendency to bioaccumulate through food chain) and essential nutrient screen (FFS Report, Appendix D, Appendix 2, Section 4.1.1 and Attachment 2) (Louis Berger et al. 2014a).
- A focused list of COPECs was identified "as [those] comprising the largest contribution of total risk to ecological receptors and were selected for evaluation in the BERA" (FFS Report, Appendix D, Section 4.1.1) (Louis Berger

et al. 2014a). These included COPECs with screening-level HQs > 100 for inorganic COPECs and > 1,000 for organic COPECs.

- The eight COPECs identified and selected for evaluation in the OU2 BERA focused on those chemicals with the greatest potential risk to ecological receptors and pathways. Specifically, the OU2 BERA evaluated the following COPECs, as they were anticipated to present the greatest risks to ecological receptors (i.e., different species of animals that could be impacted): 2,3,7,8-TCDD, copper, lead, mercury, polycyclic aromatic hydrocarbons (PAHs),[3] total DDx,[4] dieldrin, and total polychlorinated biphenyls (PCBs) (FFS Report, Appendix D, Section 4.1.1.) (Louis Berger et al. 2014a).

4.1.2 The OU2 BERA used appropriate assessment endpoints, receptor groups, and exposure pathways

- One of the hallmarks of a BERA is the use of assessment endpoints that are specific to ecological receptors or receptor groups and exposure pathways to evaluate ecological risk from contaminants at Superfund sites. The OU2 BERA used appropriate assessment endpoints that were consistent with USEPA ERA guidance for Superfund, as well as other appropriate and applicable USEPA risk assessment guidance, guidelines, and policies (FFS Report, Appendix D, Sections 4.1.3 and 4.1.4.) (Louis Berger et al. 2014a).
- The assessment endpoint used in the OU2 BERA was the protection and maintenance (i.e., survival, growth, and reproduction) of various receptor groups: benthic macroinvertebrate community, fish, aquatic-feeding birds, and piscivorous mammals (Louis Berger et al. 2014a, Appendix D).
- The OU2 BERA (Louis Berger et al. 2014a, Appendix D) assessment endpoints were consistent with USEPA guidance (USEPA 1999) because they were "ecologically relevant to the site (i.e., important to sustaining the ecological structure and function of the local populations, communities and habitats present at or near the site) and include species that are exposed to and sensitive to site-related contaminants."
- The OU2 BERA (Louis Berger et al. 2014a, Appendix D) assessment endpoints were consistent with USEPA guidance because they focused the risk assessment on "particular components of the ecosystem that could be adversely affected by contaminants from the site" (USEPA 1997).
- For the evaluation of assessment endpoints in a BERA, exposure pathways linking receptors to contaminants are evaluated. The OU2 BERA evaluated

[3] In the ERAs, PAHs were separated into high and low molecular weight PAH groups.

[4] Total DDx is the sum of all six dichlorodiphenyltrichloroethane (DDT)-related compounds (2,4′-dichlorodiphenyldichloroethane [DDD], 4,4′-DDD, 2,4′-dichlorodiphenyldichloroethylene [DDE], 4,4′-DDE, 2,4′-DDT and 4,4′-DDT).

exposure pathways of direct contact with and ingestion of contaminated sediment and biological tissue (FFS Report, Appendix D, Section 4.1.3) (Louis Berger et al. 2014a). These exposure pathways were also evaluated in the OU4 BERA (Windward 2019). Additional pathways associated with less potential for exposure and risk relative to evaluated contaminated sediment and biological tissue exposure via other pathways (e.g., surface water exposure) were also evaluated in the OU4 BERA.

- The OU2 BERA focused on those receptor groups most exposed to contaminated sediment and those with the greatest potential for risk: "the focus of this BERA is on sediment-borne contaminants in the FFS Study Area and the incidental ingestion of contaminated sediment and, due to the propensity of most of the COPECs selected to bioaccumulate, biological tissue that has accumulated contaminants through association with those sediments" (FFS Report, Appendix D, Section 4.1.3) (Louis Berger et al. 2014a).

4.1.3 The OU2 BERA used TRVs developed based on methods and analyses consistent with USEPA guidance

BERAs calculate site-specific risks based on toxicity reference values (TRVs)—the concentrations or doses above which ecologically relevant effects may occur and below which effects can be reasonably expected not to occur—to derive risk levels and inform cleanup goals at Superfund sites. The OU2 BERA (Louis Berger et al. 2014a, Appendix D) developed TRVs based on methods and analyses consistent with USEPA guidance (USEPA 1997).

- The OU2 BERA used three types of TRVs: sediment benchmarks, critical body residues (CBRs), and dose-based TRVs (FFS Report, Appendix D, Section 4.2) (Louis Berger et al. 2014a).
- Sediment benchmarks for the evaluation of benthic invertebrates were based primarily on one of the following:

  1) T20/T50 values derived from logistic regression modeling of paired sediment and toxicity data for marine amphipods[5]

  2) National Oceanic and Atmospheric Administration effects range–low and effects range–median values

  Using off-the-shelf thresholds as a line of evidence (LOE) is a common practice for evaluating benthic communities in sediment. The OU2 BERA's (Louis Berger et al. 2014a, Appendix D) reliance on sediment evaluation for benthic

[5] T20/T50 values are chemical concentrations corresponding to 20 and 50% probability, respectively, of observed toxicity. Toxicity data are based on 10-day survival of *Rhepoxynius abronius* and *Ampelisca abdita*.

community analysis was consistent with standard practice and USEPA guidance (USEPA 1999).

- The OU2 BERA (Louis Berger et al. 2014a, Appendix D) used a range of TRVs, including a lower bound (e.g., smaller percentile from a distribution of effect concentrations or no-observed-adverse-effect limit [NOAEL] based) and an upper bound (e.g., greater percentile from a distribution of effect concentrations or lowest-observed-adverse-effect limit [LOAEL]-based). The use of lower and upper bound thresholds is consistent with USEPA guidelines (USEPA 1997).
- Because USEPA guidance does not provide explicit effect thresholds to be used to calculate risks to ecological receptors, the OU2 BERA developed protective TRVs from published toxicity studies using best professional judgment (Louis Berger et al. 2014a, Appendix D). Specifically, the OU2 BERA derived TRVs from a consensus-based review process conducted by the Partner Agencies[6] (FFS Report, Appendix D, Attachment 1.3), consistent with USEPA (1998): "Because of the uncertainty in predicting the effects of biological stressors such as introduced species, professional-judgment approaches are commonly used." In addition, the use of data from scientific laboratory studies is consistent with USEPA (1998) guidelines: "Risks to organisms in field situations are best estimated from studies at the site of interest. However, such data are not always available. Frequently, risk assessors must extrapolate from laboratory toxicity test data to field effects."
- Effect thresholds used in the OU2 BERA generally are conservative, as acknowledged in the uncertainty section: "Use of the most sensitive species to select CBRs likely resulted in an overestimate of risks for the residue-based analysis." And ultimately, "the use of sensitive species to evaluate risks is appropriate and used at other Superfund Sites (USEPA, 1995c; 2003b)." The OU2 BERA further states: "Although the conservative procedures employed in the selection of CBRs tended to result in risks being overestimated, suitable tissue residue data for certain COPECs were limited and may not have included relevant sensitive species or life stages" (FFS Report, Appendix D, Section 4.5) (Louis Berger et al. 2014a).
- Some OU2 BERA CBRs/TRVs were determined through the use of extrapolation factors (EFs), whereby thresholds reported in toxicity studies were reduced (e.g., by a factor of 2, 5, or 10) to account for factors in the studies that were different than the assessment endpoints being protected (e.g., to protect more sensitive species, represent a chronic duration, or determine a no-effect level from an effect level). The use of EFs is within USEPA guidelines

[6] USEPA formed a partnership with United States Army Corps of Engineers (USACE), the State of New Jersey, the National Oceanic and Atmospheric Administration (NOAA) and U.S. Fish and Wildlife Service (USFWS) (collectively referred to as "the Partner Agencies").

and practices as stated in USEPA (1998): "Analysis phase activities may suggest additional extrapolation needs. If a stressor persists for an extended time, it may be necessary to extrapolate short-term responses over a longer exposure period, and population-level effects may become more important …. extrapolations between two species may be more credible if factors such as similarities in food preferences, body mass, physiology, and seasonal behavior (e.g., mating and migration habits) are considered (Sample et al., 1996)."

4.1.4 The OU2 BERA considered relevant site-specific data

- The OU2 BERA appropriately considered and used site-specific data. (FFS Report, Appendix D, Attachments 1.1 and 1.2) (Louis Berger et al. 2014a). Specifically, the OU2 BERA evaluated site-specific sediment, blue crab tissue, and fish tissue chemistry data used to develop exposure point concentrations (EPCs) (FFS Report, Appendix D, Section 4.2.2, Table 4 1), consistent with USEPA guidance: "Site specific data should be collected and used, wherever practicable, to determine whether or not site releases present unacceptable risks and to develop quantitative cleanup levels that are protective. Site-specific information can include, but is not limited to, plant and animal tissue residue data, toxicity test data, bioavailability factors, and population- or community-level effects studies (USEPA 1999)."
- The OU2 BERA also evaluated previously collected data on benthic macroinvertebrate community structure (FFS Report, Appendix D, Section 4.1.5) (Louis Berger et al. 2014a).
- The OU2 BERA (Louis Berger et al. 2014a, Appendix D) did not include benthic toxicity and benthic community data from OU2 that were collected as part of data collection from OU4. However, the OU2 BERA was within standard practice and USEPA guidance in relying on sediment evaluation for benthic community analysis. USEPA (1999) guidance encourages the use of site-specific toxicity tests, but it does not require such tests for the evaluation of baseline risk estimates: "The baseline risk assessment **may** include site-specific toxicity tests with test organisms that address the endpoints selected for the site. Through the use of field studies and/or toxicity tests, several types of data may be developed to provide supporting information for a lines-of-evidence approach to characterizing site risks. This approach is far superior to using single studies or tests or measurements to determine whether or not the observed or predicted risk is unacceptable (USEPA 1999)" (emphasis added).
- The OU2 BERA's (Louis Berger et al. 2014a, Appendix D) use of site-specific data, as well as its evaluation of the exposure pathways and assessment endpoints, are consistent with CERCLA's NCP (40 CFR § 300.340 (d)(4); Louis Berger et al. 2014a, Appendix D, Attachment 1.1 & 1.2 & Section 4.1.3)

4.2 CONCLUSION 2: THE ALLOCATION USED A RISK-BASED ALLOCATION METHODOLOGY.

The Allocation Recommendation Report (AlterEcho 2020) relied on the risk-based methodology underlying the OU2 BERA, which was consistent with Site data.

4.2.1 The Allocation Recommendation Report relied on the same risk-based methodology as the OU2 BERA

- The OU2 BERA strongly supported the conclusion that ecological receptors (i.e., macroinvertebrates, fish, birds, and mammals) residing and foraging in the FFS Area of the LPR are being adversely impacted as a result of exposures to COPECs (FFS Report, Appendix D, Section 6.2.) (Louis Berger et al. 2014a). Because risk is the driver for determining whether remedial actions are necessary, and because risk is used to develop PRGs that drive cleanup decisions for a site (following USEPA guidance (USEPA 1991)), it is logical to use risk-based methodologies to allocate responsibility for remedial action costs.
- The Allocation Recommendation Report (AlterEcho 2020) focused on the COPECs (also referred to as COCs) identified in the OU2 BERA: TCDD, copper, lead, mercury, PAHs (evaluated as low-molecular-weight polycyclic aromatic hydrocarbons [LPAHs] and high-molecular-weight polycyclic aromatic hydrocarbons [HPAHs]), DDx, dieldrin, and PCBs (Louis Berger et al. 2014a, Appendix D).
- The Allocation Recommendation Report's (AlterEcho 2020) risk-based methodology determined a relative ranking of the eight COCs, because these chemicals posed site-specific ecological risks in OU2. For allocation calculations, all COCs were evaluated for each ecological receptor.
- In the OU2 BERA (Louis Berger et al. 2014a, Appendix D), a subset of COPECs were found to contribute the highest risks to ecological receptors. These findings were relied upon in the Allocation Recommendation Report (AlterEcho 2020) with respect to TCDD's relative contribution to overall ecological risk, in comparison to contributions from the other seven COCs evaluated.
- Background was considered in assigning relative risk rankings from COCs in the Allocation, consistent with USEPA guidance. The evaluation of background concentrations should be included in the risk characterization section, per USEPA guidance: "Specifically, the COPCs with high background concentrations should be discussed in the risk characterization, and if data are available, the contribution of background to site concentrations should be distinguished (USEPA 2002)."

- An evaluation of background was provided in the OU4 BERA (Windward 2019) following risk characterization (and identification of COCs). This evaluation helped focus the list of ecological risk drivers for consideration by risk managers. Background was also coinsidered in the evaluation of benthic sediment quality triad data presented in the OU4 BERA.
- The Allocation Recommendation Report (AlterEcho 2020) accounted for background in determining relative risk numbers. Accounting for the contribution from background, consistent with and as required by USEPA guidance (USEPA 2002), the relative contribution of TCDD to ecological risk in OU2 averaged across all ecological lines of evidence was 77.52% (Allocation Recommendation Report, Attachment H: Allocation Protocol) (Table 1).
- The Allocation Recommendation Report's (AlterEcho 2020) assignment of 83.92% of overall relative environmental risk (cancer, non-cancer, and ecological) to TCDD was consistent with the ecological risks posed by TCDD in comparison to the risks posed by the other seven COCs considered in the Allocation Recommendation Report and identified in the OU2 ROD (USEPA 2016) and OU2 BERA (Louis Berger et al. 2014a, Appendix D) (Table 1).

Table 1. Aggregated OU2 percent contributions to overall environmental harm

COC	Incremental Percent Contribution			
	Ecological Harm	Non-cancer Hazard	Cancer Risk	Overall Environmental Harm
TCDD	77.52%	81.99%	92.24%	83.92%
Total PCBs	14.42%	16.79%	7.40%	12.87%
Total DDx	3.45%	0.55%	0.10%	1.37%
Mercury	2.23%	0.63%	0%	0.95%
Copper	2.07%	0%	0%	0.69%
Dieldrin	0.09%	0.03%	0.26%	0.13%
HPAHs	0.15%	0%	0%	0.05%
Lead	0.03%	0%	0%	0.01%
LPAHs	0.04%	0%	0%	0.01%

Source: Allocation Recommendation Report, Attachment H: Allocation Protocol, Table 3 (AlterEcho 2020).

COC – contaminant of concern
DDD – dichlorodiphenyldichloroethane
DDE – dichlorodiphenyldichloroethylene
DDT – dichlorodiphenyltrichloroethane
HPAH – high-molecular-weight polycyclic aromatic hydrocarbon
LPAH – low-molecular-weight polycyclic aromatic hydrocarbon
OU – Operable Unit
PCB – polychlorinated biphenyl
total DDX – sum of all six DDT isomers (2,4′-DDD, 4,4′-DDD, 2,4′-DDE, 4,4′-DDE, 2,4′-DDT and 4,4′-DDT)
TCDD – tetrachlorodibenzo-*p*-dioxin

4.2.2 The Allocation Recommendation Report used a balanced and practical methodology to determine relative ecological risk

- The Allocation Recommendation Report (AlterEcho 2020) took into account the relative contribution of each COC to total site risk and treated the seven ecological receptor/LOEs equally, consistent with USEPA ERA guidance and precedence for the evaluation of multiple key trophic levels as receptors. Each ecological receptor contributes value to the overall health of the ecological community (Hodgson et al. 2019).
- The Allocation Recommendation Report (AlterEcho 2020) methodology, which weighted ecological risk equally with cancer risk and non-cancer hazard in calculating each COC's relative contribution to overall risk, was consistent with risk management decision-making at Superfund sites as mandated by USEPA risk assessment guidance and the NCP (USEPA 1989, 1991).

4.3 CONCLUSION 3: THE METHODOLOGIES AND OUTCOMES OF THE OU2 BERA ARE GENERALLY CONSISTENT WITH THOSE OF THE OU4 BERA FOR ALL RECEPTOR GROUPS, WITH THE EXCEPTION OF RISK TO THE BENTHIC COMMUNITY.

The OU4 BERA (Windward 2019) built upon work performed in the OU2 BERA (Louis Berger et al. 2014a, Appendix D), with a full characterization of chemical exposures undergone by all categories of aquatic and aquatic-dependent organisms across all 17.4 miles of the LPR.

- The assessment endpoints (i.e., survival, growth, and reproduction) of receptor groups in the OU2 BERA (i.e., benthic macroinvertebrate community, fish, aquatic-feeding birds, and piscivorous mammals) (Louis Berger et al. 2014a, Appendix D) were consistent with those evaluated in the OU4 BERA (Windward 2019).
- The OU4 BERA (Windward 2019) used the same TRVs to derive risk calculations for macroinvertebrates, fish, and wildlife that were used in the OU2 BERA (Louis Berger et al. 2014a, Appendix D).
- The LOEs used in the OU2 BERA (Louis Berger et al. 2014a, Appendix D) for macroinvertebrates, fish, and wildlife were consistent with those used in the OU4 BERA: tissue-residue and dietary exposure (Windward 2019).

- Some technical aspects of risk evaluation and assumptions differed between the OU2 BERA (Louis Berger et al. 2014a, Appendix D) and the OU4 BERA (Windward 2019). This is because the OU2 BERA utilized simpler methodologies to characterize risk[7] to efficiently evaluate remedial options for OU2, whereas the OU4 BERA presented a robust and comprehensive characterization of specific receptor species (e.g., sediment-probing birds), range of potential exposure assumptions, range of effects thresholds (TRVs), and exposure pathways (e.g., surface water exposure), as well as more specific methods of evaluation (e.g., more spatial evaluation). Both the simplified methodologies presented in the OU2 BERA and the more technically robust methodologies presented in the OU4 BERA are within the parameters outlined in USEPA guidance and standard practice.
- Although the OU4 BERA (Windward 2019) was a more technically robust evaluation than the OU2 BERA (Louis Berger et al. 2014a, Appendix D), the conclusions of the two BERAs were generally consistent with each other. A longer list of preliminary COCs (including other metals, tributyltin and cyanide) with LOAEL HQs > 1.0 was identified in the OU4 BERA than in the OU2 BERA, because a more comprehensive list of receptors and additional LOEs for methods of evaluation were considered in the OU4 BERA. However, the OU4 BERA ultimately focused on a short list of ecological risk drivers—a subset of the list of COCs identified in the OU2 BERA—all of which had site-wide spatial scales of risk: TEQ (dioxins/furans, PCB, and total TEQ), total PCBs, and DDx. Mercury and copper were not included in the final risk driver list for the LPRSA BERA.
- For the benthic invertebrate community receptor group, the OU2 BERA (Louis Berger et al. 2014a, Appendix D) and OU4 BERA (Windward 2019) both evaluated tissue. However, the OU2 BERA evaluated sediment chemistry only (i.e., derived HQs for benthic invertebrates-based sediment thresholds for COPECs), whereas the OU4 BERA evaluated sediment chemistry, toxicity, and benthic community data as part of a sediment quality triad evaluation. The difference in approaches resulted in risks to the benthic community being better defined in the OU4 BERA.

[7] Technical aspects that were generalized using more simplistic assumptions in the OU2 BERA included: the use of direct sediment chemistry to the use of a "generic fish," whereby all fish species (other than mummichog) were included to generate a site-wide EPC, and the use of generic assumptions for predicting early life stage concentrations in fish and bird eggs.

4.4 CONCLUSION 4: THE ECOLOGICAL RISKS IN OU2 ARE SIMILAR TO THOSE IN OU4, AND IT IS REASONABLE TO APPLY THE SAME RISK-BASED METHODOLOGY.

- OU2 and OU4 are contiguous segments of the LPR and as such, share similar ecosystems, exposure pathways, receptors, and ecological risk drivers.
- OU2 and OU4 share a similar suite of contaminants of concern. In both the OU2 BERA (Louis Berger et al. 2014a, Appendix D) and OU4 BERA (Windward 2019), a subset of COPECs contributed the highest risks to ecological receptors. The COPECs with the highest allocations (primarily TCDD and PCBs but including DDx as the next-ranked COC for allocation percent; see Table 1) were the same as the ecological risk drivers identified in the OU4 BERA.
- The ERAs for OU2 and OU4 both used exposure pathways of direct contact with and ingestion of both contaminated sediment and biological tissue, which were the pathways associated with the most relative exposure and ecological risk.
- The ERAs for OU2 and OU4 used the same receptor groups: benthic macroinvertebrate communities, benthic invertivorous fish, piscivorous or semi piscivorous fish, aquatic-feeding birds, and piscivorous mammals.
- The upper Passaic River represents a source of background contaminants for both OU2 and OU4. Therefore, incremental adjustments of risk in OU4 to account for off-site contaminant contributions to receptor risk would be consistent with the risk adjustments used in the Allocation Recommendation Report (AlterEcho 2020).
- The conclusions relied upon in the Allocation Recommendation Report (AlterEcho 2020) with respect to TCDD's relative contribution to overall ecological risk in comparison to contributions from the seven other COCs evaluated in OU2 (Table 1) are consistent with risk estimates found in the OU4 BERA (Windward 2019). TCDD contributes the most ecological risk in both OU2 and OU4. To demonstrate the relative contribution of TCDD to overall incremental ecological risk to the contributions from the other COCs, I conducted a preliminary, exploratory analysis comparing OU2 and OU4 incremental HQs. Figure 1 shows the OU2 incremental HQs presented in the Allocation Recommendation Report (AlterEcho 2020) compared to OU4 HQs for a subset of the same receptor groups assuming the same methods and background concentration values used in the allocation methodology. Figure 1 reflects an initial analysis of incremental HQs for OU4.

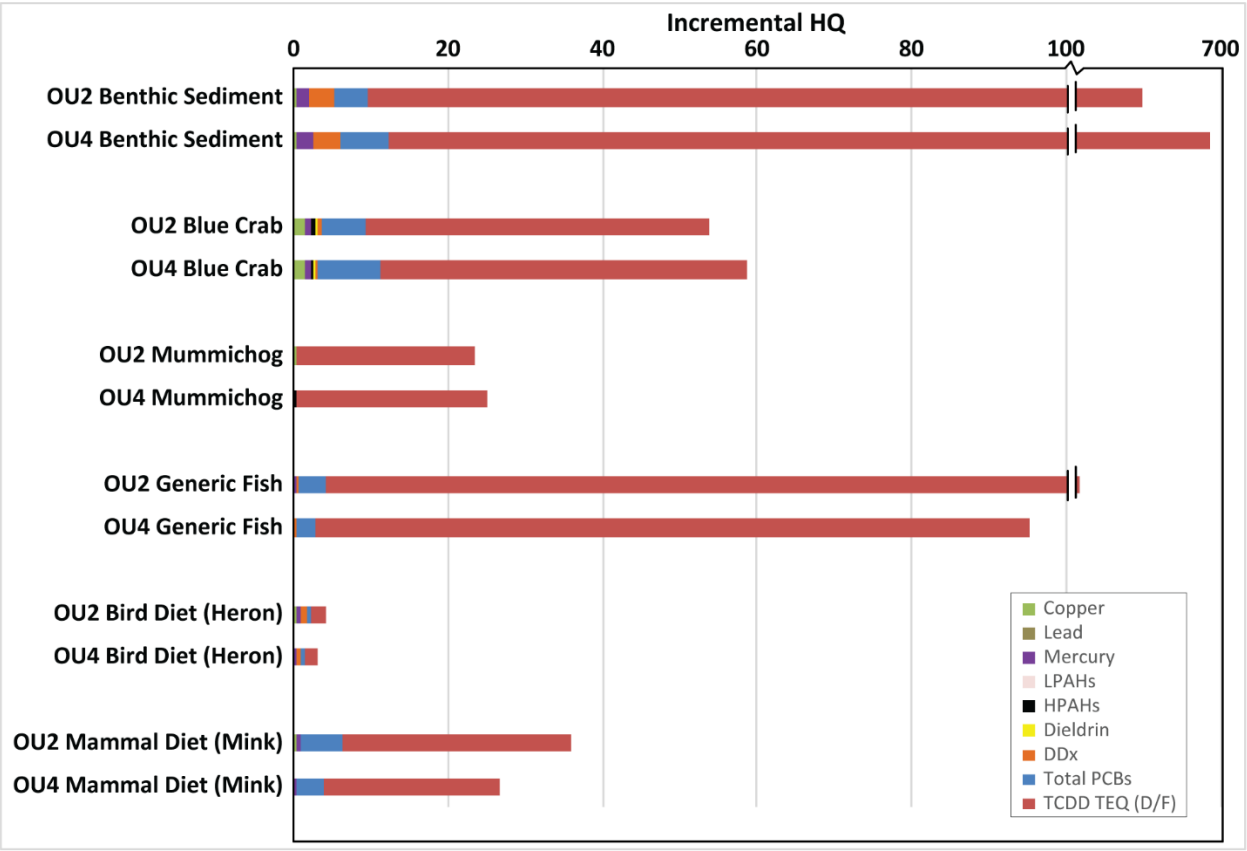

Figure 1. Comparison of OU2 incremental HQs with OU4 HQs

Notes: OU4 Incremental HQs were calculated using the same background concentration values presented in the Allocation Recommendation Report (AlterEcho 2020). These OU4 incremental HQs represent initial values for OU4.

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Exhibit C

AECOM

Relative Risks of OU2 Contaminants of Concern and Comparison with OU4

A summary of data and information regarding relative human health risks for the Lower Passaic River

Prepared by:

Betsy Ruffle
AECOM Technical Services, Inc.

Ruffle Betsy

Digitally signed by Ruffle, Betsy
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Reason: I agree to the specified portions of this document
Date: 2023.03.22 18:10:33 -04'00'

March 22, 2023

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Acronyms

BERA – Baseline ecological risk assessment
BHHRA – Baseline human health risk assessment
COC – Chemical of concern
COPC – Chemical of potential concern
CPG – Cooperating Parties Group
CSF – Cancer slope factor
DDD - Dichlorodiphenyldichloroethane
DDE - Dichlorodiphenyldichloroethylene
DDT - Dichlorodiphenyltrichloroethane
DDx - sum of 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT isomers
EPA – Environmental Protection Agency
FFS – Focused feasibility study
HEAST – Health Effects Assessment Summary Tables
HHRA – Human health risk assessment
IRIS – Integrated Risk Information System
LPR – Lower Passaic River
LPRSA – Lower Passaic River Study Area
NCP – National Contingency Plan
OU2 – Operable Unit 2 (the Lower-8 Miles of the Lower Passaic River)
OU4 – Operable Unit 4 (the 17-Mile Lower Passaic River Study Area)
PAH – Polynuclear aromatic hydrocarbon
PCB – Polychlorinated biphenyls
PCB-TEQ – Polychlorinated biphenyl toxicity equivalents
PPRTV - Provisional Peer Reviewed Toxicity Value
RfD – Reference dose
ROD – Record of Decision
TCDD – 2,3,7,8-Tetrachlorodibenzo-p-dioxin
TCDD-TEQ – 2,3,7,8-Tetrachlorodibenzo-p-dioxin toxicity equivalents
TEF – Toxicity equivalency factor
UPR – Upper Passaic River
WHO – World Health Organization

1. Professional Qualifications

I am Betsy Ruffle, Senior Managing Scientist and Vice President at AECOM Technical Services, Inc., an engineering firm providing environment, water, transportation, and infrastructure consulting services. I have 30 years of experience specializing in chemical risk assessment and toxicology, with much of my work focusing on assessing human health risks of contaminants in the environment. My responsibilities have included managing teams of scientists at both the project and departmental level. I serve as a technical expert in the firm's Risk Assessment and Contaminated Sediment practice areas and routinely provide in-house and external client strategic consultation on site-related risks, liability issues, and remedy selection.

For the past 30 years, I have led the conduct of human health risk assessments associated with chemical exposures at many Comprehensive Environmental Response, Compensation, and Liability Act ("CERCLA" or Superfund) sites and state-led sites with multiple exposure pathways, often involving rivers and other contaminated sediment sites. In my work, I have frequently evaluated exposures and risks from human consumption of fish containing bioaccumulative chemicals, including dioxin and polychlorinated biphenyls ("PCBs").

Beginning in approximately 2007, I was retained by the Cooperating Parties Group ("CPG") —a group of potentially responsible parties that the United States Environmental Protection Agency ("EPA") asked to perform the remedial investigation and feasibility study for Operable Unit 4 ("OU4") of the Diamond Alkali Superfund Site—to perform a baseline human health risk assessment ("BHHRA") for the lower 17-miles of the Passaic River (the "17-Mile BHHRA"). I was the lead author of the human health risk assessment planning documents and the 17-Mile BHHRA (AECOM 2017).

The 17-Mile BHHRA encompassed OU4, which includes both Operable Unit 2 (OU2) (the lower eight miles of the Passaic River) and the upper nine miles of the Lower Passaic River Study Area ("LPRSA"). Using site data collected by the CPG during the 17-Mile remedial investigation, the risks to human receptors who may contact contaminants in sediment or surface water or consume fish or crab from the lower 17 miles of the river were calculated. The 17-Mile BHHRA was approved by EPA in July 2017.

I have worked on other contaminated sediment sites, including Portland Harbor in Oregon, Berry's Creek in New Jersey, and the Anacostia River in the District of Columbia, to name a few. At the Portland Harbor Superfund Site, I co-led the post-Record of Decision ("ROD") pre-remedial design and baseline conditions investigation, which entailed designing and implementing multi-media sampling, including sediment, surface water, and fish tissue, and updating the risk assessment conducted during the remedial investigation. For the Berry's Creek Study Area, I serve as subject matter expert on risk and fish tissue metrics for the remedial design. For a site on the Anacostia River, I led the human health risk assessment on behalf of a potentially responsible party identified by the District's Department of Energy and the Environment. Through my work at these and other sites, I have extensive experience evaluating the health risks from exposure to contaminants through consumption of fish and other biota.

I have presented the results of my work at scientific conferences, including talks, expert panels, and posters at the bi-annual International Conference on Remediation of Contaminated Sediments, the annual meeting of the Society of Environmental Toxicology and Chemistry, and the annual International Conference on Soils, Sediments, Water, and Energy, and have served as session co-chair at several of these conferences. I served as a subject matter expert and organizer for the Sediment Management Work Group / U.S. Army Corps of Engineers Research and Develop Center's 2018 Workshop on Fish Exposure Processes at Contaminated Sediment Sites and spearheaded follow-on research on behalf of the Work Group. I am an active member of the Society of Environmental Toxicology and Chemistry and have published in peer reviewed journals, including *Integrated Environmental Assessment and Management, Environmental Research*, and *Human and Ecological Risk Assessment.* I have a bachelor's degree in Biology from Vassar College and a master's degree in Environmental Health Engineering from Tufts University.

2. Report Scope and Conclusions

2.1 Scope of Review

Between May of 2017 and December of 2020, EPA sponsored and oversaw an allocation for OU2, which was performed by AlterEcho (the "Allocation"), *available at* semspub.epa.gov/src/document/02/609904N. Underlying the Allocation's risk-based methodology was the human health risk assessment ("Lower 8-Mile HHRA" (LBG 2014a)) and ecological risk assessment that EPA previously performed for the Focused Feasibility Study ("FFS") (LBG 2014c) and the March 3, 2016, ROD for OU2 (USEPA 2016). The Allocation's risk-based methodology determined a relative ranking of eight contaminants of concern[1] ("COCs") that EPA identified in the OU2 ROD as posing the greatest risks to human health and the environment in the Lower 8-Miles.

I reviewed the Diamond Alkali Superfund Site OU2 Allocation Recommendation Report (AlterEcho 2020) and relevant documents. This report sets forth a summary of conclusions, based on my review of the site data and information available to EPA, along with my education and experience on prior studies, regarding the relative human health risks associated with the eight COCs identified in the ROD for OU2 and considered in the Allocation. My findings and conclusions focus on the *Methodology for Determination of the Relative Rank of OU2 Contaminants of Concern for the Purpose of the Allocation,* which is Attachment A to the Allocation Protocol (Attachment H of the Allocation Recommendation Report).

2.2 Summary of Conclusions

Conclusion 1. The risk assessment assumptions and approaches underpinning the Allocation's risk-based methodology are consistent with EPA guidance and standard industry practice.

Conclusion 2. The chemicals and exposure pathways included in the Allocation provide a reasonable basis for determining percent contribution to human health risk for OU2.

Conclusion 3. The toxicity factors used by EPA to calculate the health risks associated with each COC are consistent with guidance and precedent.

Conclusion 4. The Allocation methodology appropriately accounted for the background contribution to site risk.

Conclusion 5. The Allocation used a reasonable and practical methodology for determining the relative risk of each COC by considering the relative contribution of each COC to total site risk and treating cancer risk and noncancer hazard equally, which is consistent with and derives from EPA risk assessment guidance and Superfund regulations—the National Contingency Plan ("NCP").

Conclusion 6. The Allocation methodology appropriately treats 2,3,7,8-Tetrachlorodibenzo-p-dioxin ("TCDD") as the principal COC driving OU2 risk and responsible for 81.99% of the total noncancer hazard and 92.24% of the total cancer risk.

Conclusion 7.The human health risks in OU2 are substantially similar to those in OU4, making it reasonable to use the same risk-based methodology to account for relative risks posed to human health by each of the eight contaminants of concern.

[1] The Allocation Recommendation Report, Attachment A to the Allocation Protocol, identifies the following COCs: copper, DDx, dieldrin, dioxins and furans, lead, mercury, polychlorinated biphenyls, and polynuclear aromatic hydrocarbons (PAHs) which are separated into high and low molecular weight PAH groups.

3. Basis of Conclusions

This section of the report provides the technical basis for the findings summarized above.

3.1 Conclusion 1

Conclusion 1. The risk assessment assumptions and approaches underpinning the Allocation's risk-based methodology are consistent with EPA guidance and standard industry practice.

The Allocation applies a science-based framework for ranking the relative contribution of the COCs to the overall human health risk posed by a site. The approach is grounded in risk assessment, which is a well-established tool for integrating metrics of chemical toxicity and receptor-specific exposure to quantify the potential for harm posed by environmental contamination. The results of human and ecological risk assessments are used in remedy decision-making at Superfund sites (USEPA 1991).

The risk assessment framework as applied in the Allocation provides a reasonable and practical methodology for determining the relative risk posed by each COC. The methodology accounts for differences in the types and concentrations of contaminants present in site media and uses well-established metrics for determining health risks. The toxicity factors selected for each COC are consistent with EPA guidance and risk assessments conducted at other Superfund sites. The potential for each COC to elicit adverse health effects was assessed in accordance with guidance and using standard equations for calculating cancer risk and noncancer hazard (USEPA 1989). Consistent with guidance for conducting Superfund risk assessments, the exposure assumptions were selected to represent reasonable maximum exposure ("RME") to site media and focused on consumption of Lower Passaic River ("LPR") fish and crab by an angler. The concentrations of COCs in fish and crab tissue were based on a comprehensive, high quality data set that has been validated in accordance with EPA requirements.

3.2 Conclusion 2

Conclusion 2. The chemicals and exposure pathways included in the Allocation provide a reasonable basis for determining percent contribution to human health risk for OU2.

EPA selected a list of contaminants for evaluation in the Lower 8-Mile HHRA, namely the following six chemicals of potential concern (COPCs):

- Dioxins and furans (or "TCDD");
- Polychlorinated biphenyls ("PCBs");
- Total DDx (sum of isomers):
  - 4,4'- Dichlorodiphenyldichloroethane ("4,4'- DDD");
  - 4,4'- Dichlorodiphenyldichloroethylene ("4,4'-DDE"); and
  - 4,4'-Dichlorodiphenyltrichloroethane ("4,4'-DDT").
- Chlordane (cis and trans isomers);
- Dieldrin; and
- Methyl mercury.

TCDD refers to the group of seventeen dioxin and furan congeners which are evaluated as 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity equivalents ("TCDD-TEQ"); TCDD-TEQ is driven by the releases of 2,3,7,8-TCDD, which is the most toxic and most dominant of the seventeen congeners (USEPA 2010). EPA considered these COPCs to be "most bioaccumulative, most persistent in the environment, and most toxic to human receptors" and to "represent the contaminants that have triggered states to issue fish and shellfish consumption advisories or bans" (p.3-7 of LBG 2014a). Except for chlordane, these chemicals were also identified as the human health COCs for the Allocation (AlterEcho 2020). Chlordane was not

included in the Allocation, presumably due to its negligible contribution to total risk in the Lower 8-Mile HHRA (0.1 percent or less).

Focusing risk assessment calculations on a subset of chemicals is consistent with standard risk assessment practice and guidance. The EPA's guidance *Selecting Exposure Routes and Contaminants of Concern by Risk-Based Screening* states, "The baseline risk assessment process can be made more efficient by focusing on dominant contaminants and routes of exposure at the earliest feasible stage." (USEPA 1993). This is conducted using a COPC selection process that typically involves comparing maximum concentrations of chemicals against health-protective screening levels, based on the premise that any risk posed by chemicals present below their respective screening levels contribute negligibly to the total site risk (USEPA 1989, 1993).

The subset of six COPCs included in the Lower 8-Mile HHRA poses over 98% of the total human health risk for the Lower 8 Miles. Using the same screening methodology that was used to select COPCs in the approved 17-Mile BHHRA, an additional 18 compounds would be identified as COPCs for fish and/or crab tissue in OU2 (**Table 1)**. Even with the inclusion of these 18 compounds in risk calculations, over 98% of the total cancer risk and noncancer hazard is posed by the subset of COPCs (**Table 2**). The remaining less than 2% includes inorganics, pesticides, and semivolatile organics, most of which each contribute less than 0.1% to total cancer risk and noncancer hazard.

The findings are the same for OU4 – EPA's list of COPCs for the Lower 8-Mile HHRA accounts for over 98% of the total human health cancer risk and noncancer hazard in the 17-Mile BHHRA (**Tables 3** and **4**). In summary, the list of COPCs selected by EPA for the Lower 8-Mile HHRA and carried forward for the Allocation provide a reasonable and representative characterization of human health risk from consumption of LPR fish and crab.

In the Lower 8-Mile HHRA and the Allocation, EPA reasonably focused on fish and crab consumption as exposure pathways. In my experience conducting risk assessments at sediment sites where bioaccumulative chemicals are COPCs, consumption of locally caught fish or shellfish (e.g., crab) drives site risk and the need for remedial action, and other routes of exposure such as direct contact with surface water or sediment, contribute negligibly to total site risk. This is consistent with the findings of many other sediment site risk assessments where consumption of locally caught fish is a relevant exposure pathway (e.g., Hudson River and Grasse River in New York, Centredale Manor/ Woonasquatuckett River in Rhode Island, and Portland Harbor in Oregon, to name a few).

This is also supported by the 17-Mile BHHRA, which found that the risks from direct contact exposures (i.e., incidental ingestion and dermal contact) with sitewide LPR surface water and sediment are within or below EPA's acceptable risk benchmarks (AECOM 2017). For direct contact with OU4 surface sediment, the cancer risk is 6×10^{-6}, which falls within EPA's acceptable risk range of 1×10^{-6} to 1×10^{-4}, and the noncancer hazard is 0.2, which falls below EPA's target hazard index of 1 (see **Tables 3** and **4**). Risks from direct contact with OU4 surface water are even lower. Over 99 percent of the total site cancer risk and noncancer hazard for the angler receptor is attributable to consumption of LPR fish and crab, and 0.58 percent or less is attributable to direct contact with sediment and surface water (**Tables 3** and **4**). Further, most of the direct contact risk and hazard is attributable to TCDD in sediment.

In summary, the relative percent contribution of COCs to total risk in OU2 or OU4 would not change appreciably if exposure routes other than fish and crab consumption or additional COPCs besides the six evaluated by EPA in the Lower-8 Mile HHRA were considered.

3.3 Conclusion 3

Conclusion 3. The toxicity factors used by EPA to calculate the health risks associated with each COC are consistent with guidance and precedent.

Two categories of adverse health effects that may occur due to exposure to chemicals are addressed in Superfund risk assessments:

1. Carcinogenic effects, which are expressed as the probability that an individual will develop cancer over a lifetime based on the exposure assumptions used in the risk assessment, and

2. Noncarcinogenic effects, which include health effects other than cancer, such as adverse impacts to the liver, central nervous system, or developmental toxicity.

The toxicity factors used to calculate the indices of "cancer risk" and "noncancer hazard" are referred to as the cancer slope factor ("CSF") and reference dose ("RfD"), respectively (USEPA 1989). In the Lower 8-Mile HHRA and the Allocation, EPA selected toxicity factors consistent with EPA guidance, including the hierarchy for selecting toxicity factors for Superfund risk assessments, as well as several risk assessments for other Superfund sites. With three exceptions, EPA obtained the toxicity factors from the Integrated Risk Information System ("IRIS"), which is EPA's top tier source in risk assessment guidance (USEPA 2003). EPA obtained the reference doses (the toxicity factor used to calculate noncancer hazard) for 4,4'-DDD and 4,4'-DDE from Provisional Peer Reviewed Toxicity Values ("PPRTVs"), which is EPA's second tier source of toxicity factors. EPA took the cancer slope factor (the toxicity factor used to calculate cancer risk) for TCDD from the Health Effects Assessment Summary Tables ("HEAST"), which is considered a third tier source, in the absence of other finalized factors.

A range of CSFs have been used by EPA and states in risk assessments where dioxin is a COPC. The Lower 8-Mile HHRA identifies several possible CSFs (see Section 3.5 of (LBG 2014a)), including the following (where a higher value indicates greater cancer potency):

Dioxin CSF $(mg/kg\text{-}day)^{-1}$	Source
770,000	California Environmental Protection Agency 2010 Public Health Goals of Chemicals in Drinking Water (CalEPA 2010)
156,000	USEPA's Office of Health and Environmental Assessment (EPA 1985)
150,000	USEPA's Health Effects Assessment Summary Tables (EPA 1997)
130,000	CalEPA Office of Environmental Health Hazard Assessment (CalEPA 2011)

The HEAST CSF of 150,000 $(mg/kg\text{-}day)^{-1}$ selected by EPA is consistent with several other Superfund site risk assessments. For example, the HEAST CSF was used to evaluate TCDD risk at sediment sites within EPA Region 2, including Berry's Creek, Newark Bay, and Onondaga Lake. There is also precedent at sediment sites outside of Region 2, including Woonasquatucket River/Centredale Manor Restoration Site in Region 1, Atlantic Wood Industries Site in Region 3, Fox River in Region 5, and Lower Duwamish Waterway in Region 10. In addition, the state of New Jersey used the HEAST CSF to derive health-based criteria for dioxin in groundwater [https://www.nj.gov/dep/standards/1746-01-6_TF.pdf].

Consistent with the OU2 ROD, as well as the 17-Mile BHHRA, PCBs were evaluated in the Allocation as total PCBs (sum of all detected congeners). EPA's Lower 8-Mile HHRA evaluated PCBs separated into two groups – the twelve congeners with presumed dioxin-like toxicity, referred to as dioxin-like PCBs (or PCB-TEQ), and the remaining congeners, referred to as the sum of non-dioxin-like PCBs. The toxicity factors for TCDD were used to evaluate PCB-TEQ[2], and the toxicity factors for PCBs were used to evaluate the sum of non-dioxin-like PCBs. Based on the Lower 8-Mile HHRA findings, EPA concluded that "enhancement of dioxin-like PCBs [in fish and crab tissue] was not found" (p. 3-46 of Appendix D, LBG, 2014a) and derived preliminary remediation goals ("PRGs") for OU2 using the PCB toxicity factors only (see Appendix E, LBG 2014b). EPA correctly concluded that carrying forward separate calculations for the dioxin-like and non-dioxin-like groups of congeners was not necessary, and further determined that the Allocation should be based on total PCBs. This approach is reasonable and avoids "double-counting" that can arise when summing the risks from dioxin-like and non-dioxin-like PCB congener groups.

[2] The concentrations of PCB-TEQ in LPR fish and crab were calculated using toxicity equivalency factors (TEF) for mammals developed in 2005 by the World Health Organization ("WHO") that weight the toxicity of each of the 12 dioxin-like PCB congener to that of 2,3,7,8-TCDD (Van den Berg et al. 2006). New studies have been published since the 2005 WHO TEFs, including studies indicating that PCB-126, considered the most potent of the 12 dioxin-like PCB congeners, is less potent in humans than previously assumed. The WHO is in the process of reviewing and updating the TEFs [https://www.who.int/news-room/articles-detail/call-for-experts-who-initiative-to-update-the-2005-who-tef-for-dioxin-and-dioxin-like-compounds].

3.4 Conclusion 4

Conclusion 4. The Allocation appropriately accounted for the background contribution to site risk.

A primary objective of CERCLA risk assessments is to provide information on risks that can be effectively addressed through remedial actions (USEPA 2002). Accounting for background and reference area information during the risk assessment process informs the understanding of risks associated with site releases, as opposed to risks resulting from the presence of contaminants that migrated into the site or reflect regional conditions related to human activities (Judd et al. 2003). Several of the COPCs found in the Lower 8 Miles are anthropogenic contaminants commonly found in urban and industrial water ways (e.g., PCBs, PAHs, pesticides). EPA identified minor ongoing sources of contamination to the LPR to include non-point source runoff, tributaries, stormwater outfalls, combined sewer overflows, atmospheric deposition, whereas flow from the Upper Passaic River (UPR) above Dundee Dam was identified as a more substantial ongoing source (LBG 2014c, Data Evaluation Report #2). EPA determined that the UPR is an important contributor to the contaminant concentrations of PCBs, cadmium, copper, lead, mercury, and pesticides in the Lower 8 Miles and "represents a load that is substantive in comparison to the contributions originating downstream" (LBG 2014c, Data Evaluation Report #2, Table 3-2). For PAHs, the UPR load dominates any loading from within the Site. Conversely, for 2,3,7,8-TCDD, EPA concluded the UPR load is negligible relative to contributions originating within the Site.

In the Lower 8 Miles FFS and the ROD for the Upper 9 Miles of the LPR (USEPA 2021), EPA identified the area immediately upstream of Dundee Dam as representative of the contaminant load generated by the UPR watershed (LBG 2014c, Data Evaluation Report #2), and derived background sediment concentrations for use in the risk assessments for OU2 (see Table 3-1 of Appendix E, LBG 2014b). The Allocation's use of UPR data to characterize background risks for fish and crab consumption is consistent with the Lower 8-Mile HHRA and ROD for both OU2 and OU4.

Therefore, to properly evaluate Site toxicity and risk, the relative risk of COPCs should take into consideration the contribution of background to Site risks. EPA guidance, The Role of Background in the CERCLA Cleanup Process states:

> *"Background concentrations of hazardous substances, pollutants, and contaminants found at a site is a factor that should be considered in risk assessment and risk management … the contribution of background concentrations to risks associated with CERCLA releases may be important for refining COCs that warrant remedial action," since the "CERCLA program, generally, does not clean up to concentrations below natural or anthropogenic background levels." (USEPA 2002)*

Adjustment of Site risks to account for the contribution from background as was done in the Allocation by subtracting background risk from the Site risk for each COC provides a more accurate and reasonable characterization of each COC's contribution to the conditions that led to EPA's decision to implement remedial action in the Lower 8 Miles. This "incremental risk" represents the site-related risk that is the target of remedial action under the NCP.

3.5 Conclusion 5

Conclusion 5. The Allocation used a reasonable and practical methodology for determining the relative risk of each COC by considering the relative contribution of each COC to total site risk and treating cancer risk and noncancer hazard equally, which is consistent with and derives from EPA risk assessment guidance and Superfund regulations—the National Contingency Plan ("NCP").

The Allocation methodology of determining each COC's percent contribution by comparing the individual risk or hazard calculated for each COC to the sum of the risks or hazards for all COCs is supported by standard practice for conducting risk assessments, whereby cancer risk or noncancer hazards are summed across all COPCs and exposure pathways to yield total site risk or hazard. The contribution of each COPC to total site cancer risk and total site noncancer hazard can then be calculated to determine which COPC(s) are "risk drivers" and warrant further evaluation or remedial action.

EPA guidance states that remedial action is generally warranted when either the total site cancer risk exceeds the acceptable risk range of 1 x 10^{-6} to 1 x 10^{-4} or the site noncancer hazard index exceeds 1 (USEPA 1991). In other words, both cancer risk and noncancer hazard have equal weight under the NCP when determining whether remedial action is necessary to protect human health. In determining the overall risk ranking of each COC, the Allocation's assignment of equal weight to cancer risk and noncancer hazard is appropriate and consistent with risk management decision-making at Superfund sites as mandated by EPA risk assessment guidance and the NCP.

3.6 Conclusion 6

Conclusion 6. The Allocation methodology appropriately treats TCDD as the principal COC driving OU2 risk and responsible for 81.99% of the total noncancer hazard and 92.24% of the total cancer risk.

Of the five human health COCs included in the Allocation, TCDD was found to contribute most of the total cancer risk and noncancer hazard. After accounting for background, the incremental noncancer hazard for TCDD is 108 for fish consumption and 50 for crab consumption (rounded to whole numbers). The incremental cancer risk for TCDD is 3 x 10^{-3} for fish consumption and 1 x 10^{-3} for crab consumption. TCDD contributes approximately 75% to 96% to total incremental risk depending upon the scenario evaluated—cancer risk from fish consumption, noncancer hazard from fish consumption, cancer risk from crab consumption, and noncancer hazard from crab consumption (*see inset below showing Table 1 of Attachment A to the Allocation Protocol, which shows OU2 risks/hazards at the top and incremental risks/hazards at the bottom*). In comparison, the relative contribution of PCBs to human health risk ranges from approximately 4% to 24% across the four scenarios, and the remainder of the COCs contribute 1% or less to human health risk.

Table 1 Human Health Hazard and Risk Results for Child and Adult Angler Exposed to Lower Passaic River Study Area Fish and Crab

COC	Human Health (OU2 Risks/Hazards)									
	Non-Cancer Hazard (Child)					Cancer Risk (Adult/Child)				
	Fish Diet Hazard	% Contribution	Crab Diet Hazard	% Contribution	Noncancer Hazard % Contribution[a]	Fish Diet Risk	% Contribution	Crab Diet Risk	% Contribution	Cancer Risk % Contribution[a]
Copper	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
Lead	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
Mercury	2.76	1.50	0.79	1.29	1.40	NC	0.00	NC	0.00	0.00
LPAHs	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
HPAHs	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
Dieldrin	0.38	0.21	0.08	0.13	0.17	7.57E-05	2.10	1.54E-05	1.10	1.60
DDx	2.10	1.14	0.43	0.71	0.93	1.18E-05	0.33	2.96E-06	0.21	0.27
Total PCBs	69.00	37.55	9.99	16.36	26.95	6.81E-04	18.89	9.84E-05	7.01	12.95
TCDD TEQ (D/F)	109.52	59.60	49.79	81.51	70.56	2.84E-03	78.69	1.29E-03	91.69	85.19
Totals =	**184**	**100**	**61**	**100**	**100**	**3.61E-03**	**100**	**1.40E-03**	**100**	**100**
COC	**Human Health (Incremental Risks/Hazards)**									
	Non-Cancer Hazard (Child)					**Cancer Risk (Adult/Child)**				
	Fish Diet Hazard	% Contribution	Crab Diet Hazard	% Contribution	Noncancer Hazard % Contribution[a]	Fish Diet Risk	% Contribution	Crab Diet Risk	% Contribution	Cancer Risk % Contribution[a]
Copper	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
Lead	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
Mercury	0.62	0.43	0.46	0.84	0.63	NC	0.00	NC	0.00	0.00
LPAHs	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
HPAHs	--	0.00	--	0.00	0.00	--	0.00	--	0.00	0.00
Dieldrin	Bkgd>LPRSA	0.00	0.04	0.06	0.03	Bkgd>LPRSA	0.00	6.96E-06	0.52	0.26
DDx	0.53	0.37	0.41	0.74	0.55	1.58E-06	0.05	2.10E-06	0.16	0.10
Total PCBs	35.18	24.40	5.11	9.19	16.79	3.47E-04	11.05	5.04E-05	3.75	7.40
TCDD TEQ (D/F)	107.85	74.80	49.61	89.17	81.99	2.80E-03	88.90	1.28E-03	95.57	92.24
Totals =	**144**	**100**	**56**	**100**	**100**	**3.14E-03**	**100**	**1.34E-03**	**100**	**100**

Footnotes:
a = Average of fish and crab diet risks/hazards
b = Average percent contributions for noncancer and cancer human LOEs

Looking only at noncancer hazard and taking the average across the two receptor scenarios evaluated (fish consumption and crab consumption), TCDD contributes 81.99% of the total incremental noncancer hazard in OU2. Looking only at cancer risk and the average of fish and crab consumption, TCDD contributes 92.24% of the total incremental cancer risk in OU2. The dominant contribution of TCDD is expected, given its elevated concentrations in OU2 media and greater toxicity relative to other COCs. Of the COCs included in the Lower 8-Mile HHRA and Allocation, the toxicity of dioxin is by far the greatest, with toxicity factors that are thousands of times more stringent than those of the other COCs.

3.7 Conclusion 7

Conclusion 7. The human health risks in OU2 are substantially similar to those in OU4, making it reasonable to use the same risk-based methodology to account for relative risks posed to human health by each of the eight contaminants of concern.

The Allocation's relative risk ranking of COCs in the Lower 8-Miles (OU2) is consistent with the findings of the 17-Mile BHHRA (OU4). Applying the same risk-based methodology to OU4, including the use of the UPR for determining background contribution, the relative risk ranking of the COCs in the Allocation is substantially similar to the risk ranking of those COCs in OU4 (**Figure 1**). For cancer risk and non-cancer hazard, whether by fish or crab consumption, TCDD consistently ranks highest among the COCs for contribution to human health risk.

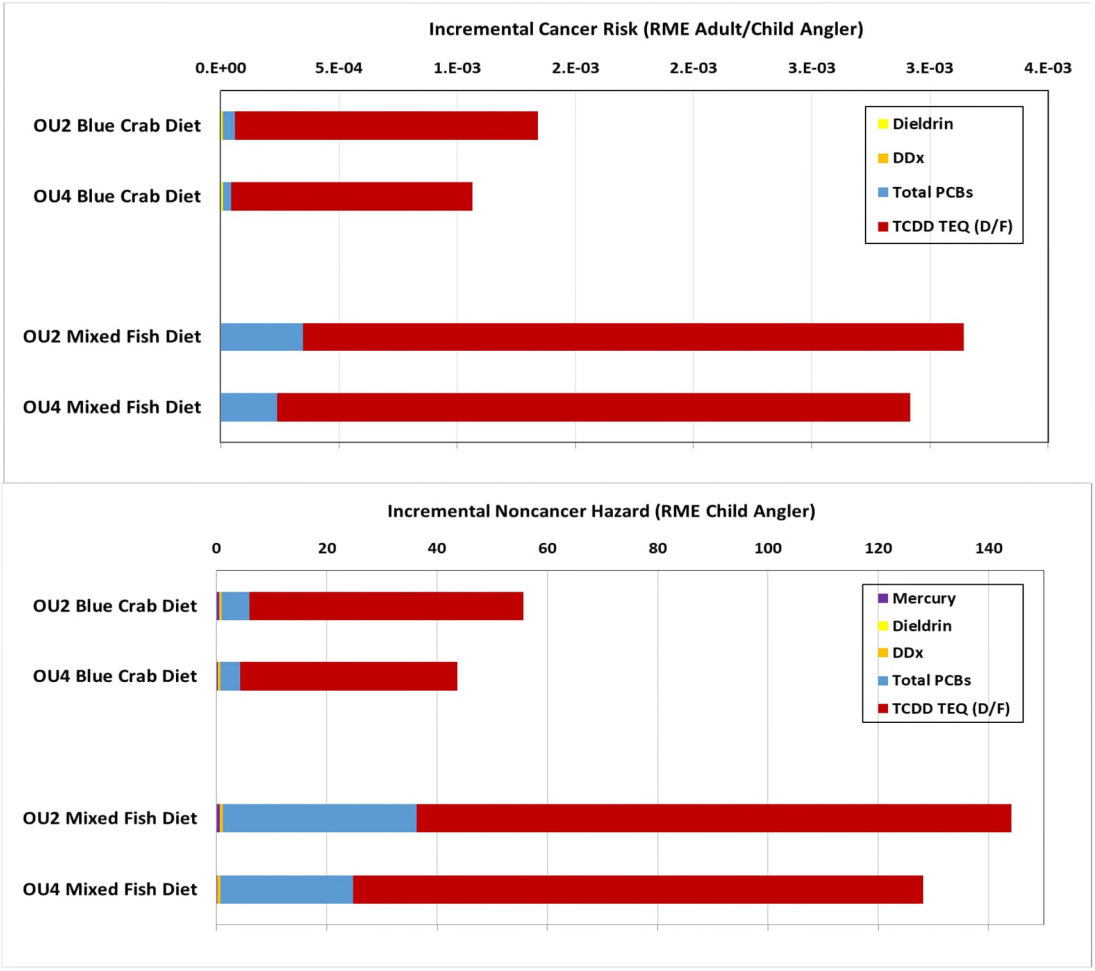

Figure 1. Comparison of Incremental Risks and Hazards for OU2 and OU4

Other human health risk factors for the two OUs are also substantially similar. For example, the suite of contaminants in OU2 is similar to that of OU4, and both OUs share the following COCs: TCDD, PCBs, DDx, dieldrin, and mercury. Likewise, the risk assessments for OU2 and OU4 both identify the UPR as the background area (see Conclusion 4). Moreover, because they are contiguous segments of the LPR between Newark Bay and the Dundee Dam, OU2 and OU4 share similar ecosystems.

Additionally, the risk assessments for OU2 and OU4 evaluated substantially similar exposure pathways and human receptors. Both risk assessments evaluated an angler who consumes fish and crab from the river. The 17-Mile BHHRA evaluated a fish diet of mixed species that is substantially similar to the mixed fish diet evaluated in the Lower 8-Mile HHRA, and the same crab tissue types (blue crab muscle and hepatopancreas tissue) were evaluated in both risk assessments. Both risk assessments also relied on the same source of fish and crab tissue data that were collected during the 17-Mile remedial investigation.

Because the human health risks in OU2 are substantially similar to those in OU4, it would be reasonable to use the same risk-based methodology to account for relative risks posed to human health and attributable to each of the eight COCs.

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Table 1. Results of COPC Screen for Lower 8-Miles Fish and Crab Tissue

Chemical of Potential Concern	Fish Tissue	Crab Tissue
Included in Lower 8-Mile HHRA (a)		
TCDD-TEQ	X	X
PCBs, total	X	X
4,4'-DDD	X	X
4,4'-DDE	X	X
4,4'-DDT	X	X
Chlordane (b)	X	X
Dieldrin	X	X
Methyl Mercury	X	X
Addition from COPC Screen (c)		
Antimony	X	NCOPC
Aldrin	X	NCOPC
Arsenic (inorganic)	NCOPC	X
Benzo(a)pyrene	NCOPC	NCOPC
bis-(2-Ethylhexyl)phthalate	X	NCOPC
Cadmium	NCOPC	X
Chromium	X	X
Cobalt	X	X
Copper	NCOPC	X
Heptachlor Epoxide	X	X
Hexachlorobenzene	X	X
Inorganic mercury	X	X
Naphthalene	X	NCOPC
Oxychlordane	X	NCOPC
Selenium	X	X
Thallium	X	X
trans-Nonachlor	X	NCOPC
Zinc	NCOPC	X

Notes:

(a) COPC included in EPA's Human Health Risk Assessment (HHRA) for the Lower 8 Miles (LBG 2014).

(b) Chlordane represents sum of alpha (cis) and gamma (trans) isomers. Total Chlordane was included as a COPC in the Lower 8-Mile HHRA, but was not carried forward as a chemical of concern (COC) in the OU2 ROD (EPA 2016) or Allocation.

(c) COPC selection based on comparison of maximum detected concentration to risk-based screening level calculated for adult who consumes 54 grams/day of fish/crab (used in EPA-approved COPC screening for the 17-Mile BHHRA (AECOM 2017)). The risk-based screening levels were calculated using EPA's Regional Screening Level Calculator assuming a per compound cancer risk of 10^{-6} and hazard quotient of 0.1.

Acronyms:

COPC - Chemical of potential concern

FFS - Focused Feasibility Study of Lower 8 Miles

NCOPC - Not a chemical of potential concern; maximum below risk-based screening level

PCB - Polychlorinated biphenyls

TCDD-TEQ - 2,3,7,8-Tetrachlorodibenzo-p-dioxin toxicity equivalents

Table 2. Summary of Baseline Cancer Risk and Noncancer Hazard for Expanded COPC List for Lower 8-Miles

Chemical of Potential Concern	Lower 8-Mile Cancer Risks and Noncancer Hazards for Fish and Crab Consumption									
	Noncancer Hazard (Child)					Cancer Risk (Adult/Child)				
	Fish Diet Hazard	% Contribution	Crab Diet Hazard	% Contribution	Noncancer Hazard % Contribution (a)	Fish Diet Risk	% Contribution	Crab Diet Risk	% Contribution	Cancer Risk % Contribution (a)
TCDD-TEQ	109.52	58.77%	49.79	80.13%	69.45%	2.84E-03	78.03%	1.29E-03	90.90%	84.47%
Total PCBs (b)	69.00	37.03%	9.99	16.08%	26.55%	6.81E-04	18.73%	9.84E-05	6.95%	12.84%
4,4'-DDD	1.76	0.95%	0.34	0.55%	0.75%	3.13E-06	0.09%	6.04E-07	0.04%	0.06%
4,4'-DDE	0.33	0.18%	0.09	0.14%	0.16%	8.37E-06	0.23%	2.22E-06	0.16%	0.19%
4,4'-DDT	0.007	0.004%	0.003	0.005%	0.004%	2.96E-07	0.01%	1.32E-07	0.009%	0.009%
Chlordane	0.095	0.05%	NCOPC	0%	0.03%	4.11E-06	0.11%	NCOPC	0%	0.06%
Dieldrin	0.38	0.21%	0.08	0.13%	0.17%	7.57E-05	2.08%	1.54E-05	1.09%	1.58%
Methyl mercury	2.76	1.48%	0.79	1.27%	1.38%	NC	0%	NC	0%	0%
Total for OU2 HHRA COPCs	***184***	***98.66%***	***61***	***98.31%***	***98.48%***	***3.61E-03***	***99.28%***	***1.40E-03***	***99.14%***	***99.21%***
Antimony	0.03	0.02%	NCOPC	0%	0.008%	NC	0%	NCOPC	0%	0%
Aldrin	0.0012	0.001%	NCOPC	0%	0.0003%	1.54E-07	0.00%	NCOPC	0%	0.002%
Arsenic (inorganic)	NCOPC	0%	0.03	0.05%	0.02%	NCOPC	0%	3.16E-06	0.22%	0.11%
Benzo(a)pyrene	NCOPC	0%	NCOPC	0%	0%	NCOPC	0%	NCOPC	0%	0%
bis-(2-Ethylhexyl)phthalate	0.05	0.03%	NCOPC	0%	0.014%	3.71E-06	0.10%	NCOPC	0%	0.05%
Cadmium	NCOPC	0%	0.05	0.07%	0.04%	NCOPC	0%	NC	0%	0%
Chromium, total	0.0001	0.00005%	0.0004	0.0006%	0.0003%	NC	0%	NC	0%	0%
Cobalt	0.06	0.03%	0.07	0.11%	0.07%	NC	0%	NC	0%	0%
Copper	NCOPC	0%	0.24	0.39%	0.20%	NCOPC	0%	NC	0%	0%
Heptachlor Epoxide	0.59	0.32%	0.29	0.46%	0.39%	1.72E-05	0.47%	8.38E-06	0.59%	0.53%
Hexachlorobenzene	0.006	0.003%	0.002	0.003%	0.003%	1.74E-06	0.05%	6.26E-07	0.04%	0.05%
Mercury (inorganic)	0.20	0.11%	0.07	0.11%	0.11%	NC	0%	NC	0%	0%
Naphthalene	0.006	0.003%	NCOPC	0%	0.002%	NC	0%	NCOPC	0%	0%
Oxychlordane	0.29	0.16%	NCOPC	0%	0.08%	2.27E-06	0.06%	NCOPC	0%	0.03%
Selenium	0.11	0.06%	0.09	0.14%	0.10%	NC	0%	NC	0%	0%
Thallium	0.33	0.18%	0.15	0.25%	0.21%	NC	0%	NC	0%	0%
trans-Nonachlor	0.81	0.43%	NCOPC	0%	0.22%	1.08E-06	0.03%	NCOPC	0%	0.015%
Zinc	NCOPC	0%	0.07	0.11%	0.06%	NCOPC	0%	NC	0%	0%
Total for Additional COPCs	***2.5***	***1.34%***	***1.1***	***1.69%***	***1.52%***	***2.62E-05***	***0.72%***	***1.22E-05***	***0.86%***	***0.79%***
Total for All COPCs	**186**	**100%**	**62**	**100%**	**100%**	**3.64E-03**	**100%**	**1.42E-03**	**100%**	**100%**

Notes/Acronyms:

(a) Average of fish and crab diet risks/hazards

(b) Consistent with the Lower 8-Mile ROD (USEPA 2016) and OU2 Allocation, PCBs were evaluated as total PCBs (sum of all congeners) using the high risk and persistence cancer slope factor (CSF) to evaluate cancer risk and Aroclor 1254 reference dose (RfD) to evaluate noncancer hazard.

COPC - chemical of potential concern

DDx - sum of 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT isomers

NCOPC - Not a compound of potential concern

PCBs - polychlorinated biphenyls

TCDD-TEQ - 2,3,7,8-tetrachlorodibenzo(p)dioxin toxicity equivalence

Table 3. Summary of Fish Consumption Noncancer Hazards and Cancer Risks and Percent Contribution to Total Site Risk - 17-Mile BHHRA

	Noncarcinogenic Evaluation							Carcinogenic Evaluation						
	Noncancer Hazard (a)				Percent Contribution			Cancer Risk (b)				Percent Contribution		
Chemical	Sitewide Sediment	Surface Water	Mixed Fish Diet (c)	Total	Surface Water and Sediment	Mixed Fish Diet	Total	Sitewide Sediment	Surface Water	Mixed Fish Diet	Total	Surface Water and Sediment	Mixed Fish Diet	Total
TCDD-TEQ	1.6E-01	2.6E-02	1.0E+02	1.0E+02	0.11%	57.84%	57.95%	4.8E-06	7.9E-07	2.9E-03	2.9E-03	0.15%	77%	77.23%
PCBs, total	1.9E-02	1.6E-03	6.9E+01	6.9E+01	0.01%	38.96%	38.97%	2.2E-07	3.6E-09	7.4E-04	7.4E-04	0.006%	20%	19.79%
4,4'-DDD	NCOPC	NCOPC	1.0E-01	1.0E-01	--	0.06%	0.06%	NCOPC	NCOPC	3.2E-06	3.2E-06	--	0.1%	0.09%
4,4'-DDE	NCOPC	NCOPC	1.7E-01	1.7E-01	--	0.10%	0.10%	NCOPC	NCOPC	7.9E-06	7.9E-06	--	0.2%	0.21%
4,4'-DDT	NCOPC	NCOPC	6.9E-03	6.9E-03	--	0.004%	0.004%	NCOPC	NCOPC	3.1E-07	3.1E-07	--	0.01%	0.01%
cis-Chlordane	NCOPC	NCOPC	1.3E-01	1.3E-01	--	0.08%	0.08%	NCOPC	NCOPC	6.3E-06	6.3E-06	--	0.17%	0.17%
trans-Chlordane	NCOPC	NCOPC	3.4E-02	3.4E-02	--	0.02%	0.02%	NCOPC	NCOPC	1.6E-06	1.6E-06	--	0.04%	0.04%
Dieldrin	1.8E-05	6.4E-06	3.2E-01	3.2E-01	0.00001%	0.18%	0.18%	4.2E-09	1.5E-09	6.9E-05	6.9E-05	0.0002%	2%	1.85%
Methyl Mercury	NCOPC	NCOPC	2.2E+00	2.2E+00	--	1.22%	1.22%	NCOPC	NCOPC	NC	--	--	--	--
Total for FFS COPCs	***1.8E-01***	***2.8E-02***	***1.7E+02***	***1.7E+02***	***0.12%***	***98.46%***	***98.57%***	***5.1E-06***	***7.9E-07***	***3.7E-03***	***3.7E-03***	***0.16%***	***99.23%***	***99.39%***
Aldrin	NCOPC	NCOPC	2.9E-03	2.9E-03	--	0.002%	0.002%	NCOPC	NCOPC	4.0E-07	4.0E-07	--	0.01%	0.01%
Aluminum	2.2E-04	NCOPC	NCOPC	2.2E-04	0.0001%	--	0.0001%	NC	NCOPC	NC	--	--	--	--
Antimony	6.8E-05	3.3E-05	6.2E-02	6.2E-02	0.0001%	0.04%	0.04%	NC	NC	NC	--	--	--	--
Arsenic, total	1.2E-03	3.3E-05	NCOPC	1.3E-03	0.0007%	--	0.001%	1.6E-07	4.3E-09	NC	1.6E-07	0.004%	--	0.004%
Benzene	3.1E-09	1.4E-06	NCOPC	1.4E-06	0.000001%	--	0.000001%	2.0E-13	9.1E-11	NC	9.1E-11	0.000002%	--	0.000002%
Benzo(a)anthracene	NC	NC	NC	--	--	--	--	1.3E-08	4.5E-09	7.7E-08	9.4E-08	0.0005%	0.002%	0.003%
Benzo(a)pyrene	1.7E-03	1.4E-03	6.7E-04	3.8E-03	0.002%	0.0004%	0.002%	1.5E-07	1.2E-07	1.1E-07	3.8E-07	0.007%	0.003%	0.01%
Benzo(b)fluoranthene	NC	NC	NCOPC	--	--	--	--	1.7E-08	1.7E-08	NC	3.5E-08	0.001%	--	0.001%
Benzo(k)fluoranthene	NC	NCOPC	NCOPC	--	--	--	--	8.1E-10	NCOPC	NC	8.1E-10	0.00002%	--	0.00002%
bis-(2-Ethylhexyl)phthalate	1.4E-04	6.5E-05	9.5E-03	9.7E-03	0.0001%	0.01%	0.01%	1.1E-08	5.2E-09	7.1E-07	7.3E-07	0.0004%	0.02%	0.02%
Bromodichloromethane	NCOPC	6.8E-08	NCOPC	6.8E-08	0.00000004%	--	0.00000004%	NCOPC	2.4E-11	NC	2.4E-11	0.000001%	--	0.000001%
C2-Benzanthracene/chrysenes	1.2E-05	NCOPC	NCOPC	1.2E-05	0.00001%	--	0.00001%	1.3E-10	NCOPC	NC	1.3E-10	0.000004%	--	0.000004%
Cadmium, diet	2.8E-04	NCOPC	NCOPC	2.8E-04	0.0002%	--	0.0002%	NC	NCOPC	NC	--	--	--	--
Chloroform	NCOPC	5.8E-07	NCOPC	5.8E-07	0.0000003%	--	0.0000003%	NCOPC	NC	NC	--	--	--	--
Chromium, hexavalent	5.8E-05	NCOPC	NCOPC	5.8E-05	0.00003%	--	0.00003%	2.5E-08	NCOPC	NC	2.5E-08	0.001%	--	0.001%
Chromium, total	2.6E-06	4.5E-07	7.8E-05	8.1E-05	0.000002%	0.00004%	0.00005%	NC	NC	NC	--	--	--	--
Chrysene	NA	NCOPC	NCOPC	--	--	--	--	1.7E-10	NCOPC	NC	1.7E-10	0.000005%	--	0.000005%
cis-Nonachlor	NCOPC	NCOPC	1.2E-01	1.2E-01	--	0.07%	0.07%	NCOPC	NCOPC	1.2E-06	1.2E-06	--	0.03%	0.03%
Cobalt	5.6E-04	7.3E-06	4.4E-02	4.5E-02	0.0003%	0.02%	0.03%	NC	NC	NC	--	--	--	--
Copper	1.0E-04	NCOPC	NCOPC	1.0E-04	0.0001%	--	0.0001%	NC	NCOPC	NC	--	--	--	--
Dibenz(a,h)anthracene	NC	NC	NCOPC	--	--	--	--	1.5E-08	3.6E-08	NC	5.2E-08	0.001%	--	0.001%
Heptachlor Epoxide	NCOPC	NCOPC	5.0E-01	5.0E-01	--	0.28%	0.28%	NCOPC	NCOPC	1.6E-05	1.6E-05	--	0.42%	0.42%
Hexachlorobenzene	NCOPC	NCOPC	4.8E-03	4.8E-03	--	0.003%	0.003%	NCOPC	NCOPC	1.7E-06	1.7E-06	--	0.04%	0.04%
Indeno(1,2,3-cd)pyrene	NC	NC	NCOPC	--	--	--	--	8.7E-09	9.0E-09	NC	1.8E-08	0.0005%	--	0.0005%
Lead	(d)	(d)	NCOPC	(d)	(d)	--	(d)	(d)	(d)	NCOPC	(d)	(d)	--	(d)
Manganese, nondiet	4.3E-04	3.1E-04	NCOPC	7.3E-04	0.0004%	--	0.0004%	NC	NC	NC	--	--	--	--
Mercury, inorganic	NCOPC	NCOPC	1.7E-01	1.7E-01	--	0.10%	0.10%	NCOPC	NCOPC	NC	--	--	--	--
Naphthalene	5.7E-06	NCOPC	NCOPC	5.7E-06	0.000003%	--	0.000003%	NC	NCOPC	NC	--	--	--	--
Oxychlordane	NCOPC	NCOPC	1.3E-01	1.3E-01	--	0.07%	0.07%	NCOPC	NCOPC	1.1E-06	1.1E-06	--	0.03%	0.03%
Selenium	NCOPC	NCOPC	8.6E-02	8.6E-02	--	0.05%	0.05%	NCOPC	NCOPC	NC	--	--	--	--
Thallium	3.7E-04	7.6E-06	4.8E-01	4.8E-01	0.0002%	0.27%	0.27%	NC	NC	NC	--	--	--	--
TPH C19-C40	9.2E-05	NCOPC	NCOPC	9.2E-05	0.00005%	--	0.0001%	NC	NCOPC	NC	--	--	--	--
TPH C9-C18	2.9E-03	NCOPC	NCOPC	2.9E-03	0.002%	--	0.002%	NC	NCOPC	NC	--	--	--	--
trans-Nonachlor	NCOPC	NCOPC	9.0E-01	9.0E-01	--	0.51%	0.51%	NCOPC	NCOPC	1.3E-06	1.3E-06	--	0.04%	0.04%
Trichloroethene	7.3E-08	3.2E-05	NCOPC	3.3E-05	0.00002%	--	0.00002%	4.8E-13	2.1E-10	NC	2.1E-10	0.00001%	--	0.00001%
Vanadium	1.2E-04	NCOPC	NCOPC	1.2E-04	0.00007%	--	0.0001%	NC	NCOPC	NC	--	--	--	--
Total for other BHHRA COPCs	***8.3E-03***	***1.9E-03***	***2.5E+00***	***2.5E+00***	***0.01%***	***1.42%***	***1.43%***	***4.0E-07***	***2.0E-07***	***2.2E-05***	***2.3E-05***	***0.02%***	***0.60%***	***0.61%***
Total for All COPCs	**1.9E-01**	**3.0E-02**	**1.8E+02**	**1.8E+02**	**0.12%**	**99.88%**	**100%**	**5.5E-06**	**9.9E-07**	**3.7E-03**	**3.7E-03**	**0.17%**	**99.83%**	**100%**

Notes/Acronyms:

-- Not a COPC in this media.
COPC - Chemical of Potential Concern.
FFS - Focused Feasibility Study.
NC - Not Calculated. No dose-response value.
NCOPC - Not a Chemical of Potential Concern.
PCB - Polychlorinated Biphenyls.
TCDD - 2,3,7,8-Tetrachlorodibenzo-p-dioxin.
TEQ - Toxicity Equivalence.
TPH - Total Petroleum Hydrocarbons

All risk and hazard values represent the reasonable maximum exposure and are from Appendix J of the 17-Mile Baseline Human Health Risk Assessment (AECOM 2017).
(a) For non-cancer, hazards shown are for the adult age group for sediment and surface water exposures (young child angler was not evaluated in BHHRA for exposure to sediment and surface water) and young child age group for biota consumption exposure.
(b) For cancer, risks shown are for combined adult and young child age groups.
(c) Mixed fish diet comprised of 20% each of American eel, white perch, smallmouth/largemouth bass, channel catfish, and common carp.
(d) Lead was evaluated in the 17-Mile BHHRA using blood lead modeling; exposure to lead in sediment and surface water was below the acceptable risk threshold.

Table 4. Summary of Crab Consumption Noncancer Hazards and Cancer Risks and Percent Contribution to Total Site Risk - 17-Mile BHHRA

	Noncarcinogenic Evaluation							Carcinogenic Evaluation						
	Noncancer Hazard (a)				Percent Contribution			Cancer Risk (a)				Percent Contribution		
Chemical	Sitewide Sediment (a)	Surface Water (a)	Crab (b)	Total	Surface Water and Sediment	Crab	Total	Sitewide Sediment (a)	Surface Water (a)	Crab (c)	Total	Surface Water and Sediment	Crab	Total
TCDD-TEQ	1.6E-01	2.6E-02	3.5E+01	3.5E+01	0.42%	78.55%	78.97%	4.8E-06	7.9E-07	9.9E-04	9.9E-04	0.51%	89%	89.55%
PCBs, total	1.9E-02	1.6E-03	7.5E+00	7.5E+00	0.05%	16.79%	16.84%	2.2E-07	3.6E-09	8.0E-05	8.1E-05	0.020%	7%	7.27%
4,4'-DDD	NCOPC	NCOPC	1.7E-02	1.7E-02	--	0.04%	0.04%	--	--	5.5E-07	5.5E-07	--	0.05%	0.05%
4,4'-DDE	NCOPC	NCOPC	4.1E-02	4.1E-02	--	0.09%	0.09%	NCOPC	NCOPC	1.9E-06	1.9E-06	--	0.2%	0.17%
4,4'-DDT	NCOPC	NCOPC	NCOPC	--	--	--	--	NCOPC	NCOPC	NC	--	--	--	--
cis-Chlordane	NCOPC	NCOPC	NCOPC	--	--	--	--	NCOPC	NCOPC	NC	--	--	--	--
trans-Chlordane	NCOPC	NCOPC	NCOPC	--	--	--	--	NCOPC	NCOPC	NC	--	--	--	--
Dieldrin	1.8E-05	6.4E-06	7.7E-02	7.7E-02	0.00006%	0.17%	0.17%	4.2E-09	1.5E-09	1.7E-05	1.7E-05	0.0005%	1%	1.50%
Methyl Mercury	NCOPC	NCOPC	5.8E-01	5.8E-01	--	1.29%	1.29%	NCOPC	NCOPC	NC	--	--	--	--
Total for FFS COPCs	***1.8E-01***	***2.8E-02***	***4.3E+01***	***4.3E+01***	***0.47%***	***96.94%***	***97.40%***	***5.1E-06***	***7.9E-07***	***1.1E-03***	***1.1E-03***	***0.53%***	***98.01%***	***98.54%***
Aluminum	2.2E-04	NCOPC	NCOPC	2.2E-04	0.0005%	--	0.0005%	NC	NCOPC	NC	--	--	--	--
Antimony	6.8E-05	3.3E-05	NCOPC	1.0E-04	0.0002%	--	0.0002%	NC	NC	NC	--	--	--	--
Arsenic, inorganic	NCOPC	NCOPC	2.3E-02	2.3E-02	--	0.05%	0.05%	NCOPC	NCOPC	2.8E-06	2.8E-06	--	0.25%	0.25%
Arsenic, organic	NCOPC	NCOPC	2.2E-02	2.2E-02	--	0.05%	0.05%	NCOPC	NCOPC	NC	--	--	--	--
Arsenic, total	1.2E-03	3.3E-05	NCOPC	1.3E-03	0.0029%	--	0.003%	1.6E-07	4.3E-09	NC	1.6E-07	0.015%	--	0.015%
Benzene	3.1E-09	1.4E-06	NCOPC	1.4E-06	0.000003%	--	0.000003%	2.0E-13	9.1E-11	NC	9.1E-11	0.000008%	--	0.000008%
Benzo(a)anthracene	NC	NC	NC	--	--	--	--	1.3E-08	4.5E-09	6.4E-08	8.2E-08	0.0016%	0.01%	0.007%
Benzo(a)pyrene	1.7E-03	1.4E-03	2.4E-03	5.5E-03	0.007%	0.01%	0.01%	1.5E-07	1.2E-07	3.9E-07	6.6E-07	0.024%	0.04%	0.06%
Benzo(b)fluoranthene	NC	NC	NC	--	--	--	--	1.7E-08	1.7E-08	8.8E-08	1.2E-07	0.003%	0.01%	0.011%
Benzo(k)fluoranthene	NC	NCOPC	NCOPC	--	--	--	--	8.1E-10	NCOPC	NC	8.1E-10	0.00007%	--	0.00007%
bis-(2-Ethylhexyl)phthalate	1.4E-04	6.5E-05	NCOPC	2.1E-04	0.0005%	--	0.0005%	1.1E-08	5.2E-09	NC	1.7E-08	0.0015%	--	0.001%
Bromodichloromethane	NCOPC	6.8E-08	NCOPC	6.8E-08	0.00000015%	--	0.0000002%	NCOPC	2.4E-11	NC	2.4E-11	0.000002%	--	0.000002%
C2-Benzanthracene/chrysenes	1.2E-05	NCOPC	NCOPC	1.2E-05	0.00003%	--	0.00003%	1.3E-10	NCOPC	NC	1.3E-10	0.000012%	--	0.000012%
Cadmium, diet	2.8E-04	NCOPC	4.6E-02	4.6E-02	0.0006%	0.10%	0.1%	NC	NCOPC	NC	--	--	--	--
Chloroform	NCOPC	5.8E-07	NCOPC	5.8E-07	0.000001%	--	0.000001%	NCOPC	NC	NC	--	--	--	--
Chromium, hexavalent	5.8E-05	NCOPC	NCOPC	5.8E-05	0.0001%	--	0.0001%	2.5E-08	NCOPC	NC	2.5E-08	0.002%	--	0.002%
Chromium, total	2.6E-06	4.5E-07	2.7E-04	2.7E-04	0.000007%	0.001%	0.0006%	NC	NC	NC	--	--	--	--
Chrysene	NC	NCOPC	NCOPC	--	--	--	--	1.7E-10	NCOPC	NC	1.7E-10	0.000016%	--	0.00%
cis-Nonachloride	NCOPC	NCOPC	3.1E-02	3.1E-02	--	0.07%	0.07%	NCOPC	NCOPC	3.0E-07	3.0E-07	--	0.03%	0.03%
Cobalt	5.6E-04	7.3E-06	6.3E-02	6.3E-02	0.0013%	0.14%	0.14%	NC	NC	NC	--	--	--	--
Copper	1.0E-04	NCOPC	2.5E-01	2.5E-01	0.0002%	0.56%	0.56%	NC	NCOPC	NC	--	--	--	--
Dibenzo(ash)anthracene	NC	NC	NCOPC	--	--	--	--	1.5E-08	3.6E-08	NC	5.2E-08	0.005%	--	0.005%
Heptachlor Epoxide	NCOPC	NCOPC	3.2E-01	3.2E-01	--	0.71%	0.71%	NCOPC	NCOPC	1.0E-05	1.0E-05	--	0.91%	0.91%
Hexachlorobenzene	NCOPC	NCOPC	1.5E-03	1.5E-03	--	0.003%	0.003%	NCOPC	NCOPC	5.3E-07	5.3E-07	--	0.05%	0.05%
Indenol(1,2,3-cd)pyrene	NC	NC	NC	--	--	--	--	8.7E-09	9.0E-09	3.8E-08	5.5E-08	0.0016%	0.003%	0.0050%
Lead	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)	(d)
Manganese, nondate	4.3E-04	3.1E-04	NCOPC	7.3E-04	0.0016%	--	0.0016%	NC	NC	NC	--	--	--	--
Mercury, inorganic	NCOPC	NCOPC	4.1E-02	4.1E-02	--	0.09%	0.09%	NCOPC	NCOPC	NC	--	--	--	--
Naphthalene	5.7E-06	NCOPC	NCOPC	5.7E-06	0.000013%	--	0.00001%	NC	NCOPC	NC	--	--	--	--
Oxychlordane	NCOPC	NCOPC	1.6E-01	1.6E-01	--	0.37%	0.37%	NCOPC	NCOPC	1.4E-06	1.4E-06	--	0.12%	0.12%
Selenium	NCOPC	NCOPC	6.3E-02	6.3E-02	--	0.14%	0.14%	NCOPC	NCOPC	NC	--	--	--	--
Thallium	3.7E-04	7.6E-06	6.6E-02	6.7E-02	0.0008%	0.15%	0.15%	NC	NC	NC	--	--	--	--
TPH C19-C40	9.2E-05	NCOPC	NCOPC	9.2E-05	0.00021%	--	0.0002%	NC	NCOPC	NC	--	--	--	--
TPH C9-C18	2.9E-03	NCOPC	NCOPC	2.9E-03	0.006%	--	0.006%	NC	NCOPC	NC	--	--	--	--
Trichloroethene	7.3E-08	3.2E-05	NCOPC	3.3E-05	0.00007%	--	0.00007%	4.8E-13	2.1E-10	NC	2.1E-10	0.00002%	--	0.00002%
Vanadium	1.2E-04	NCOPC	NCOPC	1.2E-04	0.00028%	--	0.0003%	NC	NCOPC	NC	--	--	--	--
Zinc	NCOPC	NCOPC	5.7E-02	5.7E-02	--	0.13%	0.13%	NCOPC	NCOPC	NC	--	--	--	--
Total for other BHHRA COPCs	***8.3E-03***	***1.9E-03***	***1.1E+00***	***1.2E+00***	***0.02%***	***2.57%***	***2.60%***	***4.0E-07***	***2.0E-07***	***1.6E-05***	***1.6E-05***	***0.05%***	***1.41%***	***1.46%***
Total for All COPCs	**1.9E-01**	**3.0E-02**	**4.4E+01**	**4.5E+01**	**0.49%**	**99.51%**	**100%**	**5.5E-06**	**9.9E-07**	**1.1E-03**	**1.1E-03**	**0.58%**	**99.42%**	**100%**

Notes/Acronyms:

-- Not a COPC in this media.
COPC - Chemical of Potential Concern.
FFS - Focused Feasibility Study.
NC - Not Calculated. No dose-response value.
NCOPC - Not a Chemical of Potential Concern.
PCB - Polychlorinated Biphenyls.
TCDD - 2,3,7,8-Tetrachlorodibenzo-p-dioxin.
TEQ - Toxicity Equivalence.
TPH - Total Petroleum Hydrocarbons

All risk and hazard values represent the reasonable maximum exposure and are from Appendix J of the 17-Mile Baseline Human Health Risk Assessment (AECOM 2017).
(a) Adult age group. Young child angler is not assumed to be exposed to sediment or surface water.
(b) Young child age group.
(c) Combined adult/young child.
(d) Lead was evaluated in the 17-Mile BHHRA using blood lead modeling; exposure to lead in sediment and surface water was below the acceptable risk threshold.

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